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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 6 JULY, 1927 NUMBER 1

NASOPHARYNGEAL ATRESIA

JOHN EDMUND MACKENTY, M.D.
NEW YORK

Careful search of the literature on nasopharyngeal atresia as far back as 1890 yielded surprisingly little material and few completely reported cases. The end-results, after a sufficient period to justify conclusions, were generally wanting.

J. Guisez [1] reports three cases, omitting the end-results. Severe inflammation and ulceration in the region of the subsequent atresia are given as causes. He states that atresia may follow burns from boiling liquids, chemical caustics, diphtheria, syphilis and trauma during the removal of tonsils and adenoids.

The treatment advocated is the separation of the velum palati from the posterior pharyngeal wall and the insertion of a collar-button rubber obturator, followed later by graduated dilatation.

In an interesting paper, New and Vinson [2] mention nasopharyngeal atresia as one type of atresia occurring in the oropharynx, laryngopharynx and nasopharynx. In the oropharynx, the soft palate may be scarred down to the base of the tongue; in the laryngopharynx, the atresia is usually between the base of the epiglottis in front and the hypopharyngeal wall behind. They do not discuss nasopharyngeal atresia.

Bourgevis and Poyet [3] believe that diathermy is the solution of this difficult problem, and that this method permits the formation of scar tissue with no tendency to adhere and little to contract, as distinguished from scar tissue following incision and the galvanocautery. They claim that the application of diathermy is painless and the technic simple, and that neither the wearing of an obturator nor the use of dilatation is needed. Two case reports are given. The first patient, treated in 1914 by diathermy with graduated olives and angular knives, was seen again in 1919. Though not examined, the patient could blow his nose and seemed to have normal nasal breathing. The second patient, treated for four months (five diathermy sessions), was apparently relieved at the time. A subsequent report is not given on this case.

1. Guisez, J.: Cicatricial Stenosis of the Nasopharynx, Arch. internat. de laryngol. otol. rhinol. et broncho-esophagoscopy **31**:440 (March and April) 1911.

2. New, G. B., and Vinson, P. P.: Laryngopharyngeal Diaphragm, J. A. M. A. **76**:996 (April 9) 1921.

3. Bourgevis, H., and Poyet, G.: Treatment of Certain Obstinate Strictures of the Nose and Pharynx by Diathermy, Revue de laryngol. **43**:883 (Nov. 30) 1922.

Teets [4] asserts that syphilis plays a major rôle in the production of atresia, and admits that attempts to cure are both tedious and difficult. He advocates incision, cauterization of the raw surfaces with trichloracetic acid and daily dilatation by the surgeon for months, supplemented by dilatation by the patient several times a day at home.

Leland [5] reports a case in which the patient was treated by Nichols' method. The velum palati was adherent to the base of the tongue. Glossopharyngeal adhesions were also present, binding the tongue laterally to the posterior pharyngeal wall. As dilatation failed, he resorted to the method of Nichols. The contraction was so extensive that tracheotomy was done. Food entered the esophagus through a hole about the size of a large probe. The adhesions in the nasopharynx were dense and extensive, but not complete. A central opening large enough to admit a sound about 0.5 cm. in diameter was found near the hard palate.

An operation was performed on the right side. After three months, the openings downward and upward were sufficient to admit the index finger.

Three years later, the patient reappeared and requested that an operation be performed on the left side, not because he had any serious inconvenience in breathing or in swallowing, but because he wished to eat more and faster. The right side was only slightly contracted. The second operation was as successful as the first. Dr. Leland concludes that dilatation is of no avail. The patient was nourished for several months by rectum.

The etiology is not mentioned. It is probable that syphilis was the underlying cause, since there was extensive ulceration of the face during the active period of the disease.

Hall [6] proves, by the report of two cases, that the condition is not always severe and that some patients may be cured by a simple division of the atresia. Two children, 8 and 10 years of age, were brought to him for disease of the adenoid. As he did not suspect the true nature of the obstruction, the children were anesthetized for the removal of adenoids. An atresia was found between the soft palate and the posterior nasopharyngeal wall, which yielded readily to separation with the finger. There was considerable hemorrhage, but the end-result was satisfactory. It is evident that the adhesions, though obstructive, were thin and did not include the lower edge of the velum palati (congenital type).

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4. Teets, C. E.: Atresia Pharyngis, Homoeop. Eye, Ear & Throat J. **5**:262 (Sept.) 1899.

5. Leland, G. A.: Cicatricial Stricture of the Pharyngeal Orifice, Relieved by Plastic Operation, Boston M. J. **151**:295 (Sept. 15) 1904.

6. Hall, F. J. V.: Adhesion of Soft Palate to Nasopharynx, Brit. M. J. **1**:18, 1909.

In 1914, Wright and Smith [7] collected sixty-nine cases, fifty-eight of which were due to syphilis. Their conclusion on the etiology of this disease, based on the analysis of these sixty-nine reports, gives the order of frequency as follows: (1) syphilitic; (2) traumatic; (3) diphtheritic; (4) tuberculous; (5) congenital and (6) inflammatory. Smith advocates the application of monochloracetic acid to the cut surfaces.

Bailey [8] believes that complete occlusion is rare. In his review of the literature up to 1917, he found few articles on the subject and only three cases of congenital occlusion, two of which were reported by Hall,[6] and one by Wassermann. Bailey [8] reports one congenital case of his own, which yielded to a forcible breaking down of a completely occluding membrane in the nasopharynx. The patient, seen three years later, gave no evidence of recurrence. This author's observation on the shallow anteroposterior diameter of the pharynx in cases of long standing corresponds to my own. The diameter is more apt to be shallow when the condition begins in childhood and is probably due to arrested development.

Isaacs,[9] in 1917, reported a case in which atresia followed tonsillectomy in a syphilitic patient. He believed that the syphilis was acquired about the time the operation was done; hence, it was in an active stage. His treatment consisted of incision and the wearing of a dilator on a prosthesis attached to a tooth. He gives no information on the end-result.

Ballenger [10] believes that the great majority of epipharyngeal and hypopharyngeal occlusions are due to syphilis. He is somewhat pessimistic concerning the possibility of cure, on account of the well known tendency of syphilitic scar tissue to reform and to contract.

Curtis [11] holds the opinion that strictures of the lower pharynx are less obstinate than those in the epipharynx. He gives a detailed report on a lower pharyngeal atresia in a woman with congenital syphilis, which resisted all efforts at relief and which was finally cured by an external operation in which a large skin flap from the neck was turned into the pharynx. This paper is well worth study by any one who desires to attempt this difficult operation.

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7. Wright and Smith: Diseases of the Nose and Throat, Philadelphia, Lea & Febiger, 1914.

8. Bailey, F. W.: Occlusion of the Nasopharynx, J. Iowa M. Soc. **7**:57 (Feb.) 1917.

9. Isaacs, H. E.: Stenosis of the Nasopharynx Operation with Prosthesis, Laryngoscope **27**:885 (Dec.) 1917.

10. Ballenger, W. L.: Malformation of the Pharynx; Stenosis of the Pharynx, Diseases of Nose, Throat and Ear, ed. 5, New York, Lea & Febiger, 1925, p. 370.

11. Curtis, B. F.: Cicatricial Stricture of Pharynx Cured by Plastic Operation, Ann. Surg., February, 1901.

Axhausen [12] advocates a plastic operation through pharyngotomy, gives details of the operation and reports two cases, both in women, in which this procedure was successful. One patient had tuberculosis of the lungs and the other was suspected of having syphilis.

Nichols,[13] in the most important monograph ever written on this subject, implies that syphilis is by far the most common cause of nasopharyngeal atresia. He condemns the caustic treatment so often employed in the treatment of pharyngeal ulceration, and believes that it is a potent factor in the production of scar tissue. The great ingenuity employed in the effort to reestablish an opening has been mostly in vain, since gradual closure by contraction occurs after a period of from a few weeks to two years. He believes that all procedures have one fatal defect, which defeats the result, no matter how wide the incisions or how persistent the after-treatment by dilatation or other means. The defect has its origin in the tendency of wounds to heal from their apexes or angles toward the center. By his operation, detailed later under treatment, he attempts to defeat this tendency. He reports fourteen cases in which his method was employed, with complete success in thirteen. Twelve of the patients were syphilitic. The operation was a complete failure in a negress who had a severe type of syphilis, and whose habits and environment precluded any hope of ultimate success.

In searching the literature, I have omitted several reports which evidenced an abysmal ignorance of the principles underlying this disease and which gave no end-results. These operators uniformly used division and dilatation, and reported the patients cured in a few weeks or even less.

SYMPTOMS

Nasopharyngeal atresia produces partial or complete stoppage of nasal breathing, according to the degree of obstruction. Rarely is this complete, as there usually is, at least a small, probe-sized opening into the epipharynx. Changes in the nasal mucous membrane, due to lack of aëration and to stagnation of secretions, are frequently observed, and may result in chronic sinusitis and tubotympanic catarrh with consequent deafness. One of my patients, in whom the condition had been present from birth, lost 75 per cent of her hearing; another patient showed a loss of about 50 per cent. In the others, the hearing was affected but slightly, if at all.

12. Axhausen, G.: The Operative Treatment of Supralaryngeal Pharyngeal Stenosis by External Pharyngotomy and Consecutive Plastics, Arch. f. klin. Chir. **107**:533 (April 26) 1916.

13. Nichols, J. E. H.: The Sequelae of Syphilis in the Pharynx and Their Treatment, Tr. Am. Laryng. A. **18**:161, 1896.

Pronounced pathologic changes in the larynx, trachea and lungs are not mentioned in the literature. One of my patients had chronic bronchitis and asthma, and was much relieved by an internasal operation for a hyperplastic ethmoiditis which made itself manifest after the nasal breathing was restored.

ETIOLOGY

Nasopharyngeal atresia may be congenital or acquired. A few reports of congenital atresia were gathered from the literature, to which I have added one of my own. It is a misnomer to speak of a congenital malformation as a cicatriced stenosis, as the term should be used only to characterize the pathologic end-results of inflammation or ulceration. A child may be born with a closed nasopharynx due to some inflammatory change that may have occurred after the embryonic cycle in the nasopharynx had been properly completed. It is more than probable that this is a manifestation of congental syphilis. The condition must (at least from an academic, if not from a pathologic, standpoint) be distinguished from a true congenital nasopharyngeal atresia due to an embryonic failure in which inflammation has no part. In making the distinction, one may be accused of splitting hairs. I am convinced that such a distinction exists, and that close observation may prove its existence. In the reported cases of congenital closure, the patients were cured by the simple procedure of opening the nasopharynx. In my own case, the finger in the nasopharynx encountered a smooth, somewhat yielding, shallow vault. The peculiar, irregular feel of an inflammatory adhesion was absent. The membrane dividing the nasopharynx into an upper and a lower chamber was thin, easily broken down and did not show any tendency to reform. These characteristics are foreign to inflammatory adhesions, especially when activated by syphilis, in which the resultant scar has a vicious tendency to reform. The congenital cases may, then, be divided into those due to inflammation and those due to malformation. In one patient in my series in whom congenital syphilis was demonstrated, the obstruction being present at birth, the condition might be diagnosed as congenital syphilitic stenosis. There are many causes for the acquired condition; according to the meager statistics available, syphilis accounts for the great majority. In the order of frequency these causes are: syphilis, trauma, diphtheria, tuberculosis and simple inflammation.

It is more than probable that in rare instances there is what might be called, for want of a better term, a cicatrizing diathesis, which is a determining factor in the end-result of inflammation or trama. By this I mean that the healing process results in the overproduction of fibrous tissue; otherwise, how can the rarity of this disease be explained?

Extensive syphilitic ulceration of the pharynx and nasopharynx, trauma following operation, and other conditions, are frequently

observed without resultant atresia. I believe that it would be almost impossible to experimentally produce atresia by trauma in most persons. It is a common observation that there is a marked difference in the reaction of different persons to surgical traumatism; in some patients, healing is immediate and smooth, while in others, there is a tendency to pile up granulation tissue in the wound. This difference is observed especially after tonsillectomy. Granuloma as large as the original tonsils may appear and organize into fibrous tissue which, finally contracting, may spoil the cosmetic result and may even be the source of annoyance to the patient. In one case in which my operation for atresia was done, the whole operative area was piled high with granulations in the second week. An obstinate hemorrhage from these vascular masses necessitated transfusion for its control. The patient gave a history of severe bleeding following the traumatic attack on his tonsils, which resulted in the atresia for which I operated. The first operation done by me failed on account of this abnormal tissue reaction. The palate splitting operation, detailed under treatment, gave him a fair and permanent opening into the nasopharynx. In this patient the tonsillectomy, which resulted in atresia, could not have been performed in a worse manner; yet I do not believe that the baleful result was entirely due to the traumatism. I have seen hundreds of tonsillectomies as badly done with no such end-result.

It would seem from a few case histories in the literature and from my own observations that operations on the throat in the active stage of syphilis are more prone to give poor results, not so much in an obstructive atresia as in slow healing and troublesome subsequent contraction. May not the diathesis mentioned be an attenuated hereditary or acquired syphilis? Faulty healing has been noted in operations done during, or too often after, acute infections of the throat.

DIAGNOSIS

The diagnosis is usually evident at a glance; yet atresia has been mistaken for obstruction from adenoids and nasopharyngeal tumors. One cannot conceive how this mistake can be made, except in the congenital type in which inspection may not reveal any deformity of the palate. In congenital atresia, the obstruction is usually a diaphragm midway up the nasopharynx and hidden from inspection. A complete closure of the nasal breathing, barring acute conditions, points to one of three causes: choanal atresia, congenital or acquired nasopharyngeal atresia or nasopharyngeal neoplasm. Internasal neoplasm rarely produces a complete bilateral obstruction to nasal breathing extending over a protracted period.

PROGNOSIS

Each case must be judged by the pathologic condition and by the cause. Congenital atresia is usually curable by division and divulsion of the obstructing diaphragm. In the acquired type, there is usually a probe opening into the nasopharynx, through which the horizontal and perpendicular extent of the atresia may be estimated. The more extensive the adhesions, the worse is the prognosis. If the flap operation (detailed under treatment) is to be successful, it is essential that the mucous membrane on the posterior pharyngeal wall below the normal line of the velum palati should not be entirely destroyed. The condition is almost hopeless if there is nothing but scar tissue, if the atresia above is extensive or if the whole pharynx is contracted to the center, and especially if syphilis is the cause. A good result may be expected if the atresia is shallow, if the mucous membrane below it is partly preserved, if the pharynx has a fairly wide and normal look and if the cause is traumatic or due to a simple inflammation. As in other plastic operations in this region (e. g., harelip and cleft palate), the operation should not be performed at a season of the year in which infections occur, during the period in winter when grip is prevalent, or during the excessively warm months of summer. Since adopting this plan, my own results in plastic surgery have been greatly improved, and dangerous complications have been averted.

TREATMENT

The many procedures advocated for the cure of nasopharyngeal atresia bear witness to the inadequacy of all and to the extreme difficulty in the way of even a moderate degree of accomplishment. This is due not to the lack of either painstaking effort or ingenuity on the part of the surgeon, but to the character of the disease and to the inaccessibility of the surgical field. Here one encounters a dense, fibrous scar, generally syphilitic in origin, surrounded by soft, flexible tissue which yields without resistance to fibrous contraction. The contraction is, moreover, exerted to a central point from the periphery of a circle, as well as in a perpendicular direction. The scar that results from syphilitic ulceration has a quality of contraction all its own, and is nonexistent in the fibrous tissue following ordinary inflammation. The more this syphilitic scar tissue is molested the more obstinately it asserts its supreme contractile power growing harder and tighter after each surgical intervention.

DILATATION

The nonoperative treatment is gradual dilatation. This rarely succeeds, except in minor cases. Even in these, it is a long, tedious and painful procedure. The patient may be instructed in the use of graduated bougies, passing them as often as seems indicated. Too much zeal

in their use may defeat the object, and if carried to a point of irritation, fibrous tissue may increase in bulk by the formation of new strands. A thorough dilatation at five-to-seven-day intervals is better than mild or moderately intensive daily treatments. This principle holds here as it does in other surgical fields. In the early literature, this procedure and the following were the favorite methods of treatment.

DILATATION WITH INCISION

A curved knife is used to free the soft palate from its moorings to the nasopharyngeal wall, thus restoring the nasopharynx to its full extent in all directions. Bougies are used frequently in an effort to maintain the opening. Sometimes a soft catheter is passed through the nose and out of the mouth and tied so as to hold the palate forward.

The use of transplants of skin or mucous membrane, placed against the juxtaposed raw surfaces of the released palate, has been advocated. No cases have been brought forward attesting to the virtues of this method; hence, I believe that these reports should be stigmatized as surgical vaporings, of which there is no dearth in the literature. Roe's method of a flap taken from the cheeks and turned on a twisted pedicle up behind the soft palate has never been proved in practice.

The wearing of an obturator shaped like a collar button during the healing period, which follows the release of the soft palate, is surgically unsound. The irritation induced by this foreign body in a field wide open to infection and in which it is impossible to secure complete rest, must defeat the object and result in a closure even worse than the original one.

Nichols was the first to apprehend the true pathologic nature of this deformity and to make the first stride forward in its correction. Little has been added to the knowledge of this condition since he published his paper over thirty years ago. Here is Nichols' great contribution:

> All operations and methods have one defect in common fatal to their success and which allows the reproduction of the original adhesion. The defect is, that no matter how deep the incision has been made, or how much tissue has been excised, or how strong or persistent dilatation has been exercised, the cicatricial tissue steadily advances in the process of healing from the bottom (angles) of the cut.

This is a well known general surgical principle, but it took a genius like Nichols to see its application to the subject under discussion. He then proceeded to apply the principle. He conceived that if the outer angles of the atresia could be epithelized before the soft palate was released, closing of the opening from the angles toward the center would be checked. To accomplish this, several strands of silk thread were passed from above downward through the tissues which are to form the external angles of the nasopharynx, i.e., through the area

where the posterior surface of the soft palate comes to an acute angle as it rounds backward to form the posterior nasopharyngeal wall. The strands of silk are tied in the center, forming a loop on either side, and are moved back and forth daily until a canal slightly larger than the strands is formed and cicatrized or epithelized. The bridge of tissue between the canals and the center of the pharynx is then divided. Dilatation is employed until healing takes place.

Roe modified this method by using a large silver wire in place of silk.

A few favorable results have been reported from the use of diathermy. The pharynx is opened with a specially constructed diathermy knife. Trichloracetic acid is believed by some to prevent the adhesions from reforming. I have had no experience with either of these methods.

THE AUTHOR'S METHOD

About twelve years ago my method was published, with the results in several cases. I hoped to secure a sufficient number of cases to prove or disprove the value of the operation, but I have been only partly successful. Even in a large clinic this deformity rarely appears.

It is evident that if the posterior surface of the soft palate can be covered with epithelium, the adhesions cannot reform. In atresia, the anterior surface of the soft palate is seen to be continuous with the surface of the pharyngeal wall below. A small opening is usually present in the center, through which the nasopharynx and the extent of the adhesions can be explored by probing. In six of my cases this opening was present; in two, it was absent. When it is absent, the extent of the adhesions may be outlined with a bent probe through the inferior meati. The absence of the opening, signifying a more extensive atresia, makes the operation more difficult and the outlook less hopeful, since a larger flap is needed to cover the defect. This is too often not available.

Assuming the hole to be present, and only a partially destroyed mucous membrane on the pharyngeal wall below the palatal curve, two flaps are outlined as follows: Beginning just external to the point where the probe enters the nasopharynx, the knife is carried downward half as far again as the upward extension of the adhesion. The knife is now turned outward and carried horizontally across the posterior pharyngeal wall until it reaches the position occupied by the pharyngeal pillar, then upward and slightly outward to where the dim outline of the arch of the soft palate appears. This procedure is repeated on the opposite side. A curved needle on a Pagenstecher thread is inserted near the lower inner angle of this outline and brought out in a similar position near the lower external angle. This needle must be placed inside of the outlined flap and the tread must pass superficially under the mucous membrane between the points of entrance and emergence. The outlined flap is dissected up, with a sharp knife, curved on the flat, or with a curved scissors, care being taken to go deep enough to avoid cutting the Pagenstecher thread. The dissection is carried upward and outward into the nasopharynx until the adhesions are completely freed and the nasopharynx stands open, as it should do normally. There is now an open nasopharynx, and on either side of the median line, below the normal level of the soft palate, and forming a part of it, are two pendulous flaps, from near the lower borders of which emerge

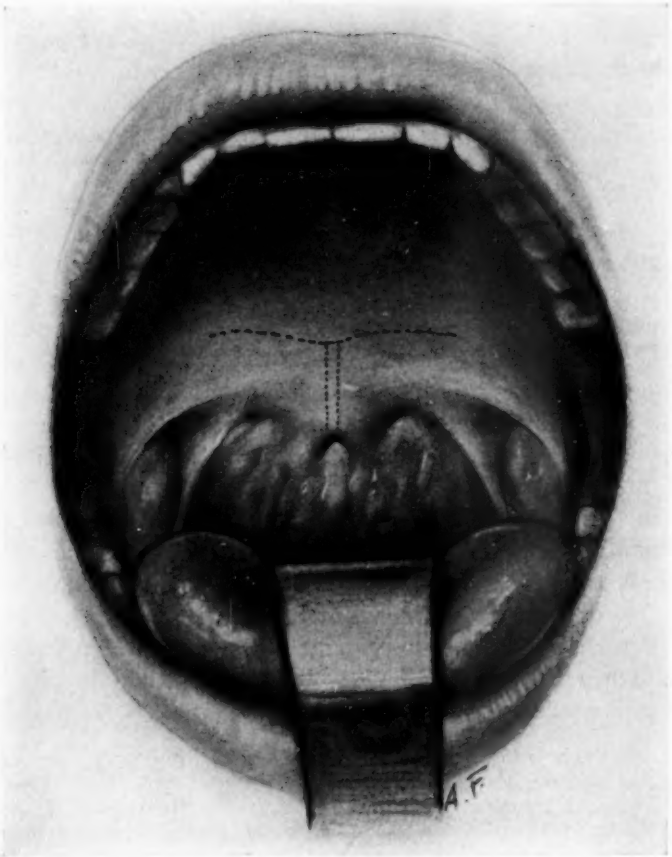

Fig. 1.—Procedure 1; soft palate adherent to posterior pharyngeal wall; probe opening into nasopharynx. The dotted lines indicate extent of atresia.

Fig. 2.—Procedure 1; probe exploring extent of atresia.

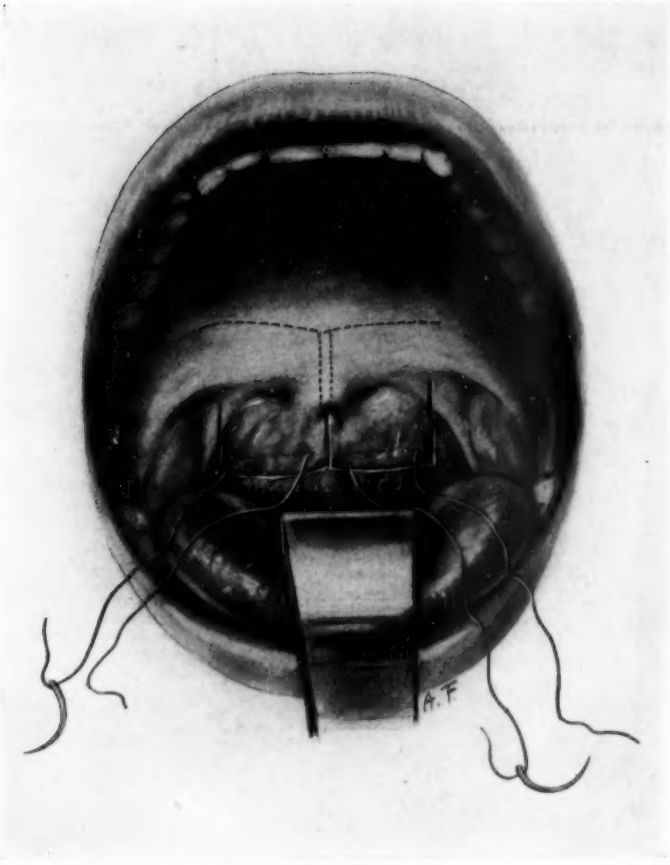

Fig. 3.—Procedure 1; flaps outlined on posterior pharyngeal wall; sutures passed through lower border of flaps.

Fig. 4.—Procedure 1; flaps dissected up; palate freed from nasopharyngeal wall.

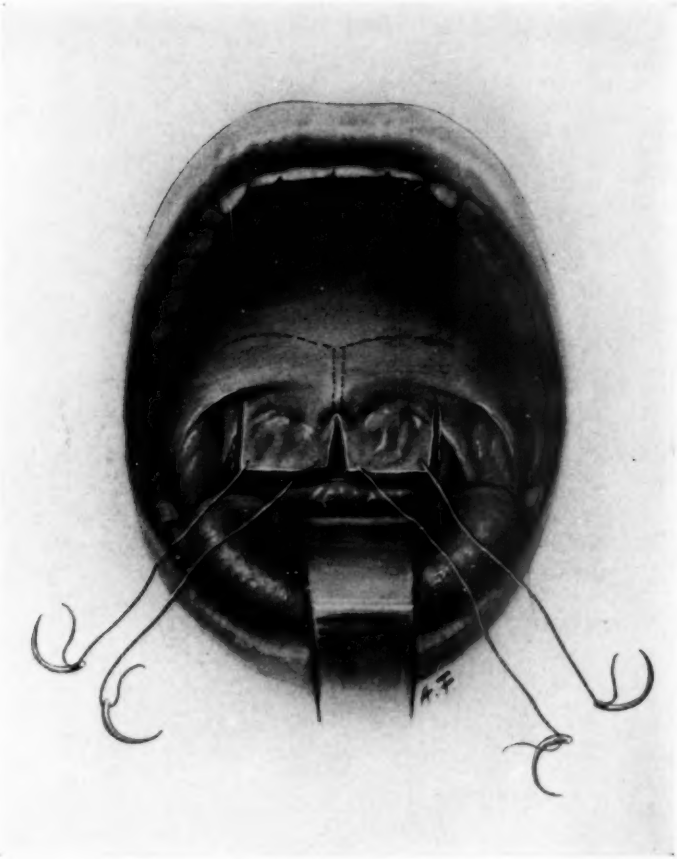

Fig. 5.—Procedure 1; flap hanging below curve of soft palate and continuous with it; needles on four ends of the sutures.

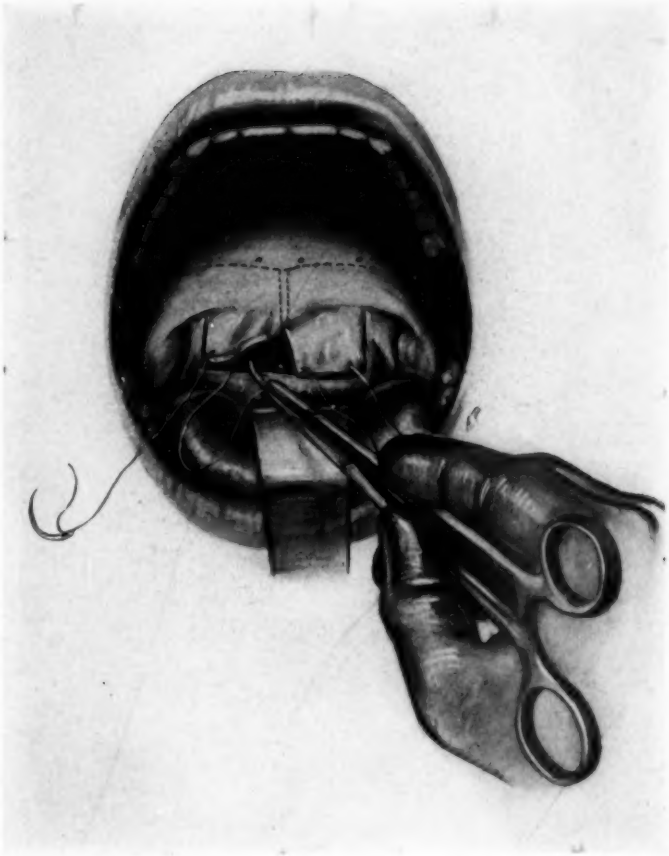

Fig. 6.—Procedure 1; method of passing curved needle behind flaps. Needles emerge at points dotted above line of atresia.

Fig. 7.—Procedure 1; sutures drawn forward causing flaps to double upward, thus applying the two raw surfaces against one another.

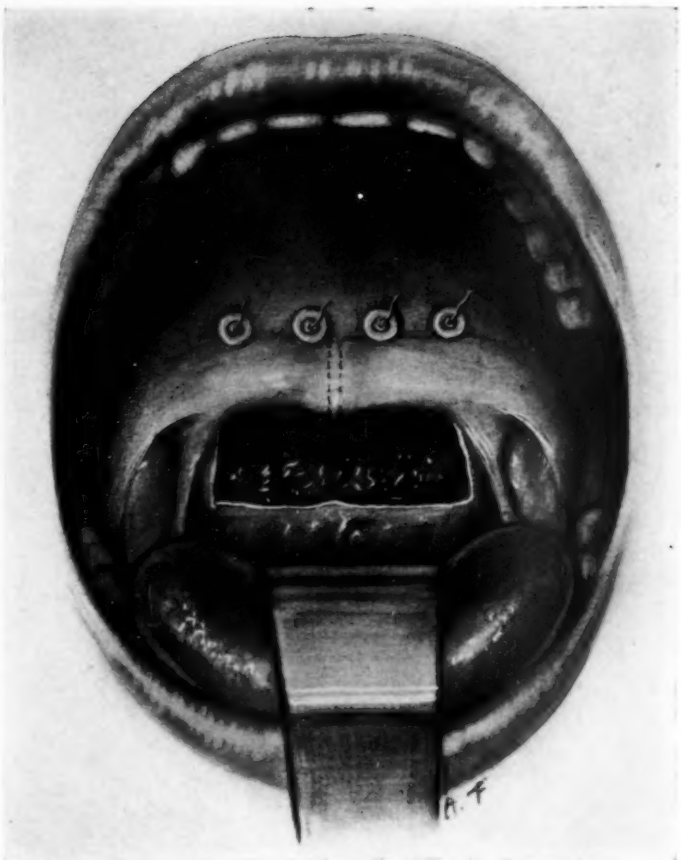

Fig. 8.—Procedure 1; sutures brought through lead disks and secured with shot.

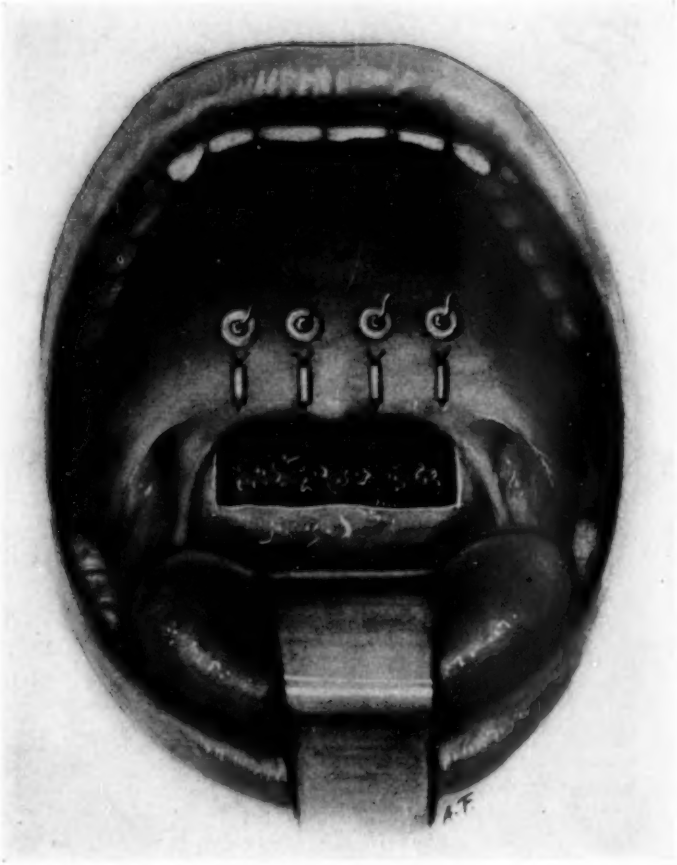

Fig. 9.—Procedure 1; other sutures applied for better coaptation of surfaces. They are passed through diminutive lead tubes to prevent cutting into palatal tissue.

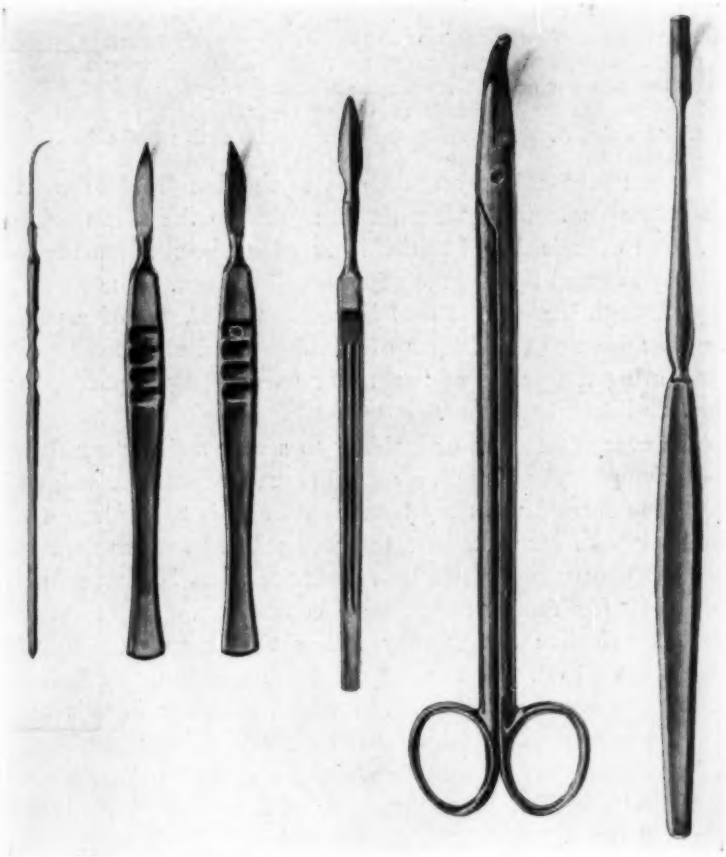

Fig. 10.—Procedure 1; instruments used in operation. The needle holder is not included.

two Pagenstecher threads. Four ends emerge from the mouth. On each is threaded a curved needle. Starting at one end of this row of threads a needle is grasped by a needle-holder in such a way that the needle is continuous with the shaft of the needle-holder, making traction on the flap through which the thread passes. The palate is drawn forward, the needle passed behind it and plunged, in an anterior direction, through the soft palate at a point above the line of atresia. The remaining three threaded needles are similarly passed and the threads drawn forward. The four threads emerge from the soft palate in a line across its base, i. e., well up toward the hard palate. Traction on the threads doubles the flaps backward and upward on themselves and applies them against the posterior surface of the palate. The mucous surfaces of the flaps face the posterior nasopharyngeal wall and the raw surfaces of the flaps are brought against the raw surface of the soft palate. Other stitches are used to secure good approximation of the parts. All are tied over lead disks and through diminutive lead tubes.

The use of these disks and tubes is to equalize pressure and to prevent the threads from sinking out of sight into the soft tissue of the palate. The threads should be tied loosely to make allowance for edema, for if they are tied tightly, the stitches will cut out. The thread, after passing through the disk, is held in position by means of a perforated shot. To minimize the danger of the disks and shot falling into the trachea during the period of healing or when being removed, the four threads are knotted together.

In the event of failure or inability to secure satisfactory flaps from the posterior pharyngeal wall, a second procedure was devised, which so far has been successful in my hands. Its aim is to produce a cleft in the soft palate as far up toward the hard palate as seems necessary for a sufficient permanent opening. Allowance must be made in making this opening for some postoperative contraction. Objection may be made that defective speech may follow the operation. In the few patients operated on, this has not occurred. Since it is of paramount importance to restore both breathway and drainage in this region, I believe one is justified in taking some chance in this regard.

A horizontal incision is made along the normal palatal curve from one pillar to the other. From the center of this, a perpendicular incision is carried through the soft palate upward well beyond the upper line of the atresia. The adhesions are freed laterally to this on both sides. The soft palate is now divided into two triangular portions. The anterior and posterior mucous membranes of these flaps is united by a continuous or interrupted stitch across their raw edges. To make this more effective and to do away with tension as far as possible, a v-shaped trough is made in the edges of the flaps, a strip of tissue being removed from the two sides of each triangle. This requires a sharp knife and an assistant to keep the tissues taut.

Dilation should be carried out after both of these operations to such an extent or for such time as seems indicated.

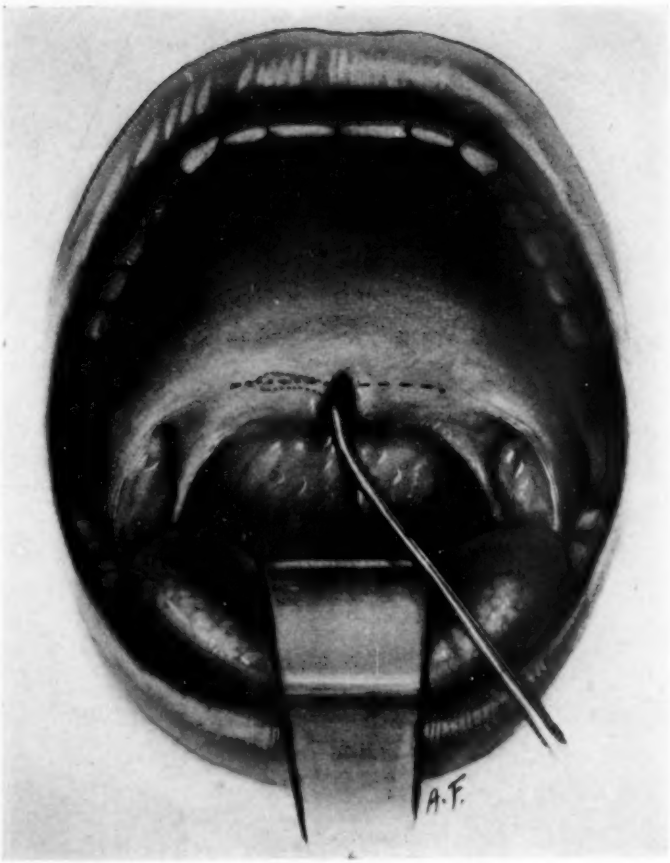

Fig. 11.—Procedure 2; probe exploring extent of atresia through small opening into nasopharynx.

Fig. 12.—Procedure 2; incisions outlined in splitting soft palate and in freeing adhesions.

Fig. 13.—Procedure 2; soft palate split, adhesions freed.

Fig. 14.—Procedure 2; posterior and anterior mucosa of soft palate seen. The thick palatal tissue is thinned by removing tissue from between the two surfaces of the mucous membrane, this relieves the tension of bringing together the two mucous membranes.

Fig. 15.—Procedure 2; anterior and posterior mucous membranes of soft palate united by continuous stitch.

Fig. 16.—Procedure 2; operation completed. The result is a partial or complete cleft of the soft palate.

SUMMARY

Ten cases have come under observation since about 1906. The first operative procedure was successful in seven and failed in three; the second procedure succeeded in establishing a sufficient opening in these three.

The cause of the atresia in the ten cases was as follows: syphilis, four; trauma caused by the removal of adenoids and tonsils, two; cause not ascertained, two; congenital arrested development, one, and probably scarlet fever, one.

BRONCHIECTASIS ASSOCIATED WITH DISEASE OF THE NASAL ACCESSORY SINUS

ETIOLOGY AND BRONCHOSCOPIC TREATMENT OF BRONCHIECTASIS *

LOUIS H. CLERF, M.D.
PHILADELPHIA

It has long been extremely discouraging to treat patients who have bronchiectasis. With the advent of bronchoscopy as a means of diagnosis and treatment of pulmonary conditions, renewed interest has been manifested in the disease. Modes of treatment have been devised and modified, and investigations along etiologic lines have been carried out to establish, if possible, other factors that might in some way influence the present methods of treatment.

Attention was directed by Mullin [1] to the frequent association of bronchiectasis and disease of the nasal accessory sinuses. He found that these conditions coexisted in practically all of his cases of bronchiectasis. In an investigation to determine whether this was a causal relationship, Mullin and Ryder [2] carried out a series of experimental studies on animals. They definitely established a lymphatic route which connects the paranasal sinuses with the bronchi and lungs, via the lymph nodes and vessels, lymphatic ducts, veins and right side of the heart. They concluded that secondary infection of the bronchi and lungs might result from disease of the nasal accessory sinuses. Another important observation made was that there was an occurrence of secondary involvement of the peritracheobronchial and peribronchial lymph nodes. Mullin and Ryder also demonstrated that there was no known direct lymphatic pathway from the lymph nodes of the neck and head to the lungs and pleura. Their ultimate conclusions were that the routes of infection are: (1) lymphatic, via the route indicated, and (2) bronchial, by inhalation of infectious material escaping into the nose from the diseased sinuses.

The influence of inspirated infectious material on the mucosa of the larynx, trachea and bronchi has been frequently observed. At the bronchoscopic clinic it is not uncommon to see patients who complain of cough and huskiness of the voice. Mirror laryngoscopy usually reveals a chronic laryngotracheitis. Thorough physical examination and roentgen-

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* From the Chevalier Jackson bronchoscopic clinics, Philadelphia.

* Read at the Annual Meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society at Brooklyn, Jan. 8, 1927.

1. Mullin, W. V.: The Lymph Drainage of the Accessory Nasal Sinuses, Tr. Am. Laryngol. Rhinol. and Otol. Soc., 1919.

2. Mullin, W. V., and Ryder, C. J.: Studies in the Lymph Drainage of the Upper Air Passages and Accessory Sinuses, Laryngoscope **31**:158 (March) 1921.

ray study of the chest fail to show any outstanding pulmonary disease. All systemic examinations are negative. Investigation of the paranasal sinuses reveals the presence of a chronic infection. In many cases a diagnostic bronchoscopy is performed, and this shows a chronic bronchitis, presumably an extension of the chronic tracheitis. Bronchial dilatation cannot be demonstrated by bronchoscopy or by pneumography; however, in a certain number of cases, changes are observed about the carina and bronchial spurs, which usually consist of a widening of the angle of division of the trachea or bronchi, the carina and spurs being broad and rounded. These changes can be interpreted as enlargement of the peritracheobronchial and peribronchial lymph nodes, as pointed out by Mullin.[3]

In referring to textbooks on laryngology one finds that chronic infection of the accessory sinus is one of the commonly accepted etiologic factors in the production of a chronic laryngotracheitis; also, that active treatment for the nasal condition will often be followed by improvement or complete cure of the infection in the lower respiratory tract. I have commonly found, by performing bronchoscopy in these cases, that a chronic bronchitis is associated with the laryngotracheitis. It may therefore be assumed that inspiration of material from an infected nasal fossa can, and does, produce a laryngotracheobronchitis, and that removal of the primary focus will do much toward clearing up the secondary infection. There is such a vast amount of clinical evidence to substantiate the relationship between disease of the paranasal sinus and chronic laryngotracheobronchitis that I believe it can be accepted as a clinical fact.

Norris and Landis [4] state that chronic bronchitis is the most important and most common predisposing factor in the production of bronchiectasis. Obviously, the length of time required for this transition cannot be expressed; however, it is probable that a considerable period must elapse before a chronic bronchitis incident to chronic disease of the nasal accessory sinus would produce bronchiectasis. This manner of production does not explain the extensive, well developed bronchiectasis associated with sinus disease which occurs in very young children who have recently had measles, whooping cough or other infectious diseases. It is likely that in the course of the primary illness, a simultaneous infection of the nasal sinuses, bronchi and probably of the lungs occurred, and that the infection in the lower respiratory tract has been perpetuated by the infection of the accessory sinus.

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3. Mullin, W. V.: The Relation of Paranasal Sinus Infection to Disease of the Lower Respiratory Tract, J. A. M. A.: **87:**739 (Sept. 4) 1926.

4. Norris, G. W., and Landis, H. R. M.: Diseases of the Chest and the Principles of Physical Diagnosis, Philadelphia, W. B. Saunders Company, 1920.

GENERAL CONSIDERATION OF BRONCHIECTASIS

Many factors must be taken into account in the consideration of non-tuberculous bronchiectasis. Etiologically it may occur as a sequel of one of the acute infectious diseases such as measles, whooping cough, scarlet fever and influenza; it may follow lobar pneumonia or bronchopneumonia; it may occur in abscess of the lung, and occasionally it is observed in bronchial foreign bodies of prolonged sojourn (Jackson). In many of these cases bronchiectasis is associated with chronic disease of the paranasal sinuses. Anatomically, it may be unilateral and monolobar or it may be bilateral and involve many lobes. The type of dilatation may be cylindrical, usually involving the larger bronchi, or sacculated, more often involving the terminal bronchioles; there may be a combination of these.

A large number of cases of bronchiectasis have been observed at the bronchoscopic clinic, and a concomitant disease of the paranasal sinus was found in many. It has been convenient to classify these cases from the standpoint of associated disease of the nasal accessory sinus because of certain differences in etiologic condition, extent of involvement and treatment.

1. *Cases of Bronchiectasis Commonly Associated with Disease of the Nasal Accessory Sinus.*—This group includes those cases in which there is a history of productive cough dating back to an infection of childhood or to an attack of influenza, particularly one contracted during the recent pandemic. There may or may not be a history of nasal symptoms; however, careful investigation of the nasal accessory sinuses will disclose some form of chronic disease, either suppurative or hyperplastic. Examination of the chest commonly shows bilateral physical signs. Bronchoscopic studies usually reveal involvement of the bronchi of both lower lobes and occasionally of the middle lobe, and pneumography, often shows that it is more extensive on one side. The bronchiectasis may be cylindric or sacculated and commonly involves the terminal bronchioles (fig. 1).

2. *Cases of Bronchiectasis Commonly Unassociated with Disease of the Nasal Accessory Sinus.*—In this group, the bronchiectasis occurs as a complication or sequel of lobar pneumonia or bronchopneumonia other than the influenzal form, abscess of the lung or bronchial foreign body of prolonged sojourn. The involvement is commonly unilateral, and usually a single lobe is involved. There may be infection of the paranasal sinus; more often there is none, even in the presence of a suppurative focus attended with profuse, purulent expectoration which often is projected into the post-nasal space during severe paroxysms of coughing (fig. 2).

Clinically the foregoing groups are usually recognized as cases of suppuration of the lungs. However, there is a group of cases of bronchiectasis in which the patients give a history of recurring colds; these cases occur more often in children. The colds develop more frequently during the winter but the patient is never completely free from cough, which is usually unproductive, and often the outstanding symptom. A history of measles, whooping cough or other disease in early childhood is often elicited. There are rarely symptoms referable to the nose. Physical examination reveals signs of a chronic bronchitis, and a diagnosis of

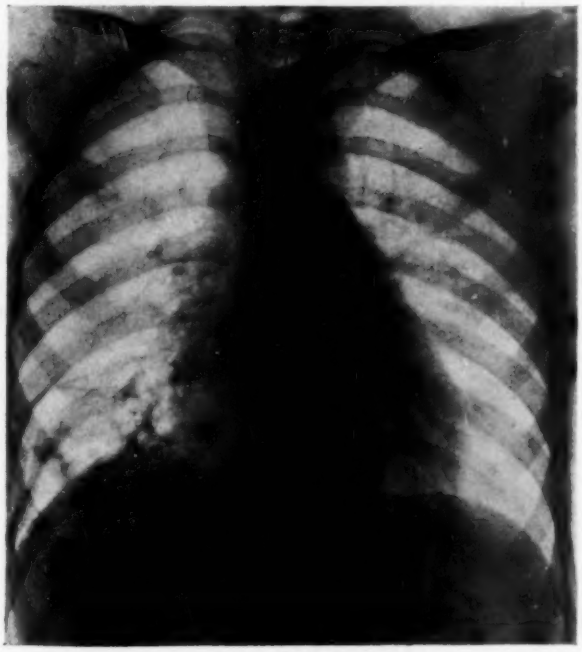

Fig. 1.—Pneumogram made after the bronchoscopic instillation of iodized oil in the right lower lobe bronchus of a child, aged 5, who at the age of 2 had contracted measles complicated by bronchopneumonia. This was followed by cough and occasional fever and later by expectoration of purulent and, at times, fetid secretion. There were no symptoms referable to the nose or nasal accessory sinuses. By bronchoscopy a chronic suppurative bronchitis was found, which involved both bronchi of the lower lobe. Pneumography demonstrated a bronchiectasis involving the right lower lobe with definite bronchiectatic dilatations of the terminal bronchi. Examination of the nasal accessory sinuses revealed a bilateral suppurative maxillary sinusitis. (Report on pneumogram by Dr. W. F. Manges.)

bronchiectasis is commonly made. The lack of expectoration is explained on the basis that children rarely expectorate. Roentgen-ray studies show chiefly heavy peribronchial shadows, principally about the hilum. If a diagnostic bronchoscopy is performed, a chronic, low grade bronchitis with scant, and at times tenacious, secretion is found. Frequently there

is widening of the carina and of the angles of division of the bronchi, the carina and spurs being thickened and rounded. There is no demonstrable bronchiectasis.

A complete and careful nasal examination commonly shows a chronic infection of the paranasal sinuses, and this furnishes the etiologic factor.[2]

Can these be considered as potential cases of bronchiectasis? If a long continued, chronic bronchitis, frequent cough and a peribronchitis, all of which tend to weaken the bronchial walls, increase intrabronchial pressure and interfere with bronchial motility and ciliary activity, and an associated chronic infection of the paranasal sinuses are to be accepted

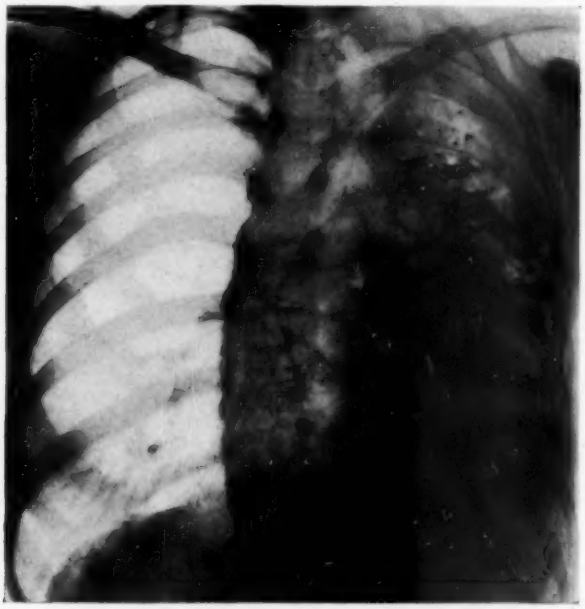

Fig. 2.—Pneumogram, made after bronchoscopic instillation of iodized oil, shows extensive bronchiectasis and pulmonary fibrosis involving the left lower lobe and a portion of the upper lobe. In this case there was a history of choking on a vegetal foreign body thirty-three years prior to presentation, when the patient was 4 years of age. Since that occurrence the patient has had a cough, periodic fever and expectoration of purulent sputum which in recent years has increased so that the quantity now varies from 6 to 8 ounces (21.3 to 28.3 Gm.) daily. During the severe paroxysms of coughing, purulent, fetid secretion would frequently be projected into the postnasal space and drain from the anterior nares. Thorough examination of the nose and nasal accessory sinuses failed to show any signs of infection. (Report on pneumogram by Dr. W. F. Manges.)

as predisposing factors to bronchiectasis, then the answer must be in the affirmative.

The following case, recently observed, demonstrates the early progress in these cases, and I am convinced that it is a counterpart of many a case in which a well defined bronchiectasis has ultimately developed.

REPORT OF A CASE

A boy, aged 10, was referred to the bronchoscopic clinic by Dr. Norman MacNeill of the pediatric out-patient department for bronchoscopic study, because of cough of six years' duration. There was a history of measles and influenza at about the fourth year. Since then, the child had never been well, as he caught colds frequently. Cough seemed to be the outstanding symptom; symptoms referable to the nose were slight and were considered as a cold. By physical examination, Dr. MacNeill found evidence of a bronchial infection. A roentgen-ray study of the chest by Dr. John T. Farrell, Jr., revealed extensive peribronchial thickening with heavy hilum shadows. The patient was referred to Dr. H. H. Lott for examination of the nose and of the nasal accessory sinuses. He reported the presence of chronic suppuration involving the sinuses, notably the maxillary antrums. At diagnostic bronchoscopy a chronic tracheobronchitis was found; a small quantity of thick purulent secretion was present in the left main bronchus; the carina was broader than normal and was rounded. There was no evidence of bronchial dilatation. Iodized oil was instilled into both lower lobes and showed a practically normal bronchial tree.

The patient is now under treatment for the sinus disease; general systemic treatment is being carried out by the pediatrician, who also advises that bronchoscopic aspiration with endobronchial medication be carried out.

The prognosis in this case should be excellent. Demonstrable bronchiectasis is not present, and the bronchial infection should be controlled if the treatment advised is carried out.

BRONCHOSCOPIC TREATMENT OF BRONCHIECTASIS

The late D. Braden Kyle, in his lecture on the influence of nasal disease in conditions of the pharynx and larynx, delivered to the undergraduate students of the Jefferson Medical College, often referred to the leaking waterpipe on the first floor as the cause of the flooded basement, and advised that prompt repair of the pipe was the first thing that should have attention. This analogy can be properly applied to cases of bronchiectasis associated with sinus disease.

All cases of bronchiectasis should be carefully studied by the internist, roentgenologist, rhinologist and bronchoscopist. Many patients do not give a history of symptoms referable to the nose, but a careful investigation shows the existence of disease of the paranasal sinus.[5]

Not every case of bronchiectasis is a suitable one for bronchoscopic treatment; however, every patient with a chronic productive cough should receive the benefit of a diagnostic bronchoscopy whenever there is a question of diagnosis, provided no contraindications to this procedure are present. Not infrequently a localized bronchiectasis has resulted from a bronchial foreign body,[6] bronchial neoplasm, bronchial stenosis or other lesions.[7] Pneumographic studies will frequently furnish addi-

5. Raffo, E.: Concomitance of Sinusitis and Bronchiectasis, Arch. Otolaryng. **2**:557 (Dec.) 1925.

6. Manges, W. F.: Pathologic Changes in Lung Tissue as the Result of Foreign Bodies of Long Sojourn, J. A. M. A. **87**:987 (Sept. 25) 1926.

7. Jackson, Chevalier: Chronic Nonspecific Infections of the Lungs, J. A. M. A. **87**:729 (Sept. 4) 1926.

tional data not obtainable in any other manner and often necessitates a reconsideration of the treatment outlined.

In cases of bronchiectasis unassociated with sinus disease, treatment should be carried out by the internist and surgeon with or without the aid of bronchoscopic aspiration, as must be determined in every case. The excellent results obtained by bronchoscopy in certain of these cases seem to warrant giving this method of treatment a definite place as an adjunct to those now in use by the internist or the surgeon.

If there is a concomitant infection of the nasal accessory sinuses, the patient should first be treated for this. If he has not been treated, the sinues infection, irrespective of its original etiologic significance, unques-

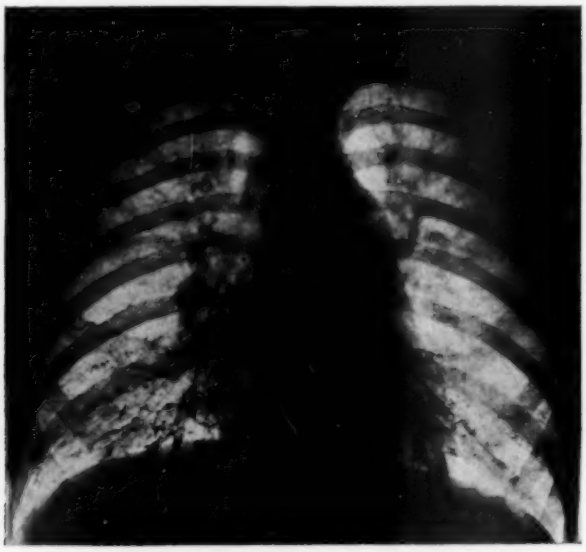

Fig. 3.—Pneumogram made in the case of a child, aged 10, following the bronchoscopic insufflation of bismuth subcarbonate into both lower lobe bronchi. There was no definite evidence of pathologic dilatation of any of the bronchi. Rhinologic examination revealed a chronic suppurative process involving the nasal accessory sinuses. A chronic suppurative bronchitis was found at bronchoscopy. Cooperation between the rhinologist, pediatrist and bronchoscopist has practically resulted in a complete cure in this case. (Report on pneumogram by Dr. John T. Farrell.)

tionably exerts an unfavorable influence on bronchiectasis. In certain cases it may be well to advise postponement of bronchoscopic aspiration. In my experience, it has been considered advisable to institute bronchoscopic treatment as soon as the sinus disease has been under control, provided bronchoscopy is to be utilized.

Chronic cough in children, often diagnosed as bronchiectasis, offers an excellent field in which brilliant results can be secured by careful investigation and prompt treatment on the part of the rhinologist and

bronchoscopist cooperating with the pediatrician. In these cases, no examination is complete until the nasal accessory sinuses have been carefully studied. With the institution of proper treatment, it is not unlikely that many cases of chronic tracheobronchitis can be cleared up before bronchiectasis develops (fig. 3).

CONCLUSION

1. Abundant clinical observations supported by experimental studies indicate that chronic disease of the nasal accessory sinus is a factor in the production of bronchiectasis. The inhalation route of infection plays an important part in cases of chronic bronchitis; in children, the peribronchitis associated with peritracheobronchial glandular involvement undoubtedly plays an important rôle.

2. Examination of a patient complaining of chronic cough, with or without expectoration, is never complete until a careful study of the nasal accessory sinuses has been carried out; a negative opinion on sinus disease should not be given until every necessary diagnostic means has been utilized.

3. The unfavorable effect of chronic sinusitis on the bronchial infection in cases of chronic bronchitis suggests a similar influence in cases of associated bronchiectasis.

4. Every patient complaining of unexplainable chronic cough or in whom a questionable diagnosis of bronchiectasis has been made, should receive the benefit of diagnostic bronchoscopic and pneumographic studies, if considered advisable.

5. Irrespective of their etiologic relationship, treatment for a coexisting bronchiectasis and suppurative sinusitis is not complete until the proper treatment is given for diseased nasal accessory sinuses and the bronchial condition.

128 South Tenth Street.

ABSCESS OF THE BRAIN DUE TO COLON BACILLUS WITH PNEUMOCEPHALUS

LYMAN RICHARDS, M.D.
BOSTON

Bernstein, in 1914, was unable to find in the literature a single reported case of abscess of the brain due to infection by the colon bacillus or any statement that this organism is ever the cause of such a lesion. His own case [1] was that of a young man, aged 24, who entered the hospital for relief of headache, a left aural discharge, chills and fever. The ear had discharged six weeks previously, but had been dry up to the onset of symptoms. Headache and vomiting increased, temperature and pulse rate became subnormal, and the patient died without having an operation performed. Lumbar puncture ten hours before death showed a clear spinal fluid high pressure, no bacteria and 79 per cent of polymorphonuclear leukocytes. The discharge from the ear showed a typical *Bacillus coli-communis.* Autopsy revealed an abscess the size of a hen's egg in the left temporosphenoidal lobe, the contents of which had a foul odor and yielded a pure culture of *B. coli.* Direct extension of infection from the pyramid or tegmen tympani could not be demonstrated, and the author considers that the infection of the brain occurred via the lymphatics from the middle ear.

In 1926, Dandy,[2] analyzing twenty-eight cases of traumatic pneumocephalus, mentions one in which an acute mastoiditis was followed by a temporosphenoidal abscess which was diagnosed by exploratory puncture. At this time thick green pus mixed with gas under great pressure was evacuated. He says: "Since the infection was presumed to be due to a gas producing organism surgical treatment (of the abscess) was considered futile." Subsequent roentgenograms showed air in the region of the abscess and also in areas corresponding to the anterior horn of the lateral ventricle without, however, any subsequent increase in its extent. Dandy concluded from this fact that the air was not formed by the organisms, which were not cultivated for the production of gas, but was forced into the cavity by coughing, sneezing or swallowing. Hence, the case was classified as one of traumatic pneumocephalus. Death occurred three days after drainage of the abscess. In discussing bacterially formed air in the brain, Dandy reports having been unable to find any case suspected or diagnosed during life, and he considers that such a discovery would be of academic interest only.

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1. Bernstein: M. Rec. **85**:249 (Feb.) 1914.

2. Dandy, Walter: Pneumocephalus (intracranial pneumatocele or aerocele), Arch. Surg. **12**:949 (May) 1926.

With the exception of these two instances, similar cases have not been found in the literature. Granted that others have occurred but have not been reported, the relative rarity of the condition warrants its publication, particularly when accompanied by such unusual complications as those in the present instance.

The question of the bacteriologic factor in abscess of the brain seems to be one about which little is said. The best and most complete monograph on the subject fails to mention this etiologic factor. This may arise from the fact that many abscesses are sterile, that the chief interest at the time of operation centers on the operative technic, and that a knowledge of the infectious organism avails little in making a decision as to subsequent therapeutic measures. This factor was of some diagnostic importance when complications appeared in my case.

REPORT OF A CASE

History.—J. L., a boy, aged 16, was admitted to the Peter Bent Brigham Hospital on Aug. 6, 1925, complaining of headache and chronic bilateral discharge from the ears. His present illness began on July 24, when he developed a severe headache which continued intermittently for two weeks. During the week previous to admission, he vomited on various occasions, and finally developed a high temperature and rapid pulse rate, for which he was brought to the hospital. He was practically unable to give any account of his symptoms, and appeared extremely ill.

Examination.—When seen in consultation, he presented the typical picture of a purulent meningitis, having a stiff neck, a double Kernig sign, a purulent spinal fluid under markedly increased pressure, with a cell count of 800 and a temperature of 103 F. Examination of the ears showed a thick, greenish-yellow discharge with a foul odor and extensive destruction of both drums and portions of the ossicles. There were no signs of an acute infection of the mastoid.

Operation and Course.—In view of the fact that a patient with almost identical, but unilateral, otitis media and with meningeal signs had been operated on and had made a speedy recovery, it was decided that some form of dural exposure and drainage was indicated. Following lumbar puncture, the patient was given 30 cc. of antimeningococcic serum, although a smear showed no organisms, and none ever grew on subsequent culture.

A bilateral mastoidectomy was performed under ether anesthesia in the hope of finding an epidural or subdural abscess that might be occasioning a localized, protective meningitis amenable to drainage. The mastoid cortex was opened on each side, a portion of the posterior canal wall taken down without any attempt at a plastic canal flap, and the mastoid antrum opened and carefully cleaned out. The overhanging bone external to the middle fossa was removed, and an area of dura, about 1.5 cm. in diameter was uncovered by removal of the antral roof, although no gross break in the latter was evident on either side. No incision was made in the dura and no exploratory puncture was made in either temporosphenoidal lobe. The latter procedure was omitted largely because in a similar case, referred to in the foregoing, a lobar puncture had been negative, and it was feared that puncture might occasion a cerebral complication, which did not exist at the time. The lateral sinus was exposed on the right side, but was found to show no evidence of thrombosis. Both wounds were lightly packed and partially closed with interrupted silk.

On the next day, August 7, the patient was in a fair condition, except for an abnormally slow pulse rate ranging between 40 and 60. There was less stiffness of the neck than before operation, although the patient was still drowsy and

complained of headache. Another lumbar puncture was made, which showed a still cloudy fluid with 1,000 cells. There was considerable relief of symptoms. Two days later, distinct improvement was noted in the general condition. The pulse rate and temperature were normal, and the stiffness in the neck had almost entirely disappeared. This was in exact accord with the results of operation in the other patient, and it was confidently expected that rapid convalescence would follow. The dressings were changed daily, and showed a continuation of discharge from each canal.

Gradually, however, it became evident that something was wrong. The patient became more irritable; he complained of intermittent headache, which was severe at times. His appetite was poor, he obviously lost weight, and his pulse rate after rising to 90, fell daily. When seen by the neurologic consultant, however, he was feeling well, and nothing but the bradycardia and a slight injection of the right optic disk pointed to what was always under consideration, a temporosphenoidal abscess. Surgical intervention was decided against at this time, in spite of the fact that with a normal temperature the white cell count was 24,000.

On August 17 a second neurologic examination revealed a definite blurring of both nasal margins of the disks and, in addition, a typical and almost complete left-sided homonymous hemianopsia. This fortunate observation settled all doubts as to diagnosis of abscess, and furnished an answer to the puzzling question as to which side the abscess occupied.

Second Operation.—The same day, under local anesthesia, the right mastoid wound was reopened, and a small incision was made in the previously exposed dura. A ventricular needle was inserted about 1.5 cm. into the right temporal lobe, following which there was an immediate dropping of purulent fluid from the end of the needle. This continued, and, aided by suction, almost 100 cc. of thick, yellow, foul-smelling pus was evacuated until blood streaks appeared, whereupon the needle was withdrawn. The momentous question of drainage next came up. It was felt that evacuation was complete, and that there was a good chance that further reaccumulation of pus would not occur; also that if signs of pressure reappeared, a second evacuation could be safely carried out through the same area, since this was well walled off from the adjacent meninges. Consequently, a simple rubber drainage wick was placed against the dura, and the wound was packed wide open.

On the next day the general appearance of the patient was considerably improved. He seemed brighter, did not complain of headache, and his pulse rate was more rapid. There were no signs of meningitis. Late in the afternoon the patient was visited by his family, who found him sitting up in bed reading and free from complaints. Two hours later, the house officer reported that the patient was practically unconscious, had a pulse rate of 45, a normal temperature and no signs of meningitis. The situation, of course, suggested a reaccumulation of pus or possibly blood in the abscess cavity, though it was hard to understand how this could have occurred in so short a time to give so much pressure.

Third Operation.—Operation was, however, distinctly indicated. Without anesthesia, a ventricular needle was again inserted through the opening in the dura, beyond which point distinct resistance was encountered, presumably offered by the capsule of the abscess. When the capsule was pierced, the abscess cavity was easily entered, and the stylet withdrawn. To the amazement of all present, no pus or blood appeared, but instead a sudden gush of foul-smelling gas was heard to escape under great pressure. A few cubic centimeters of pus was aspirated. The gas could have resulted only from fermentation due to the colon bacillus, the pus from the abscess having shown this organism to be present in pure culture at the previous operation. A small rubber catheter was inserted into the abscess cavity and retained in place with silk sutures catching it to the edges of the dura, and the wound was lightly packed with gauze. By this time the patient was able to answer questions intelligibly, and his pulse rate had risen to 68. On August 21, four days later, the patient's condition was improving steadily, and

he was better than at any time since admission. His pulse rate ranged about 60; he ate and slept well and was mentally alert. Treatment consisted in irrigations of the abscess cavity, first with boric acid and later with a surgical solution of chlorinated soda. Smears of the return fluid at two day intervals showed a gradual reduction in the number of organisms; this was also accompanied by the disappearance of the foul odor.

Convalescence continued satisfactorily; the patient was up and around on September 1, his hemianopsia disappeared, and he presented no evidence of a pathologic intracranial condition. The drainage tube gradually extruded itself, and the wound filled with clean granulations and finally closed entirely. There was still some discharge from the external canal. Strange to say, the left mastoid wound likewise healed promptly, with a cessation of the discharge from the middle ear, in spite of the fact that only a partial radical mastoid exenteration was performed, no attempt being made to do more than afford dural drainage.

The patient has now been discharged from the hospital for sixteen months and is under outpatient observation. Beyond a slight aural discharge from the right canal, he has not shown any symptoms. Whether it is advisable to complete the radical exenteration in order to eliminate this remaining symptom is still to be decided, but it would seem best not to risk stirring up the cerebral infection too soon.

COMMENT

Such a gratifying result in a condition which has so often proved fatal in spite of the most skilful treatment prompts some consideration of certain features of the case which may have had a bearing on its outcome.

Meningitis: When the patient was admitted to the hospital the signs and symptoms of meningitis were the outstanding features. Abscess of the brain was considered and dismissed as unlikely. An investigation of the nature of meningitis of otitic origin leads at once to some confusion, owing to the fact that various authors do not agree as to what constitutes a true meningitis, some including under this head only those fluids in which organisms can be grown, others regarding any case with a cell count of over 250 and a high polymorphonuclear percentage as purulent meningitis. Certain it is that many purulent spinal fluids with counts over 1,000 frequently show no organisms and are best regarded, as by Yerger, as a circumscribed reaction to local infection due to extradural or epidural abscess, abscess of the brain or pachymeningitis. This is the so-called protective meningitis which may often be interpreted as a favorable indication of local resistance and future recovery if the focus of infection can be reached and drained without converting the circumscribed meningitis into a diffuse one. Since no organisms were found in the present case, the meningitis was interpreted as being of the circumscribed type, offering hope of recovery through operation.

Operation: The success of simple dural exposure in a previous case prompted me to repeat the procedure bilaterally; I was prepared to find possibly some form of dural abscess. A local collection of pus was not found, however, and, as stated, it seemed inadvisable to explore the brain. Subsequent developments seemed to indicate that again simple dural exposure was sufficient to clear up a local protective meningitis.

One point of difference with regard to the former case was always noted, namely, that there was no postoperative reduction in the number of cells in the spinal fluid, although the clinical signs of meningitis disappeared almost entirely, only intermittent headache remaining. Several authors have called attention to the value of this sign and have urged frequent lumbar punctures for the detection of a pathologic condition which may not appear clinically. Thus the possible development of some intracranial lesion was constantly held in mind, and a lookout kept for the definite symptoms which finally made certain the diagnosis and location of the abscess.

The Abscess: In dealing intelligently with abscess of the brain, it is essential to have a clear understanding of the anatomic characteristics of this lesion which furnish such important facts concerning its location. Abscesses are classified in three main groups: (1) intracerebral, without macroscopic cortical involvement, (2) intracerebral with a cortical stalk and (3) meningeal (subdural or intra-pia-rachnoidal). The first of these arises from a retrograde thrombophlebitis, a lateral sinus thrombosis, a mastoiditis or a sinusitis. The origin of the second is like that of the first, plus some degree of direct extension from the infected focus, from which the stalk comes later to lead to the abscess. The third type results from direct extension from the contiguous focus. On this basis, the present case falls into group 2, since there was a distinctly walled off approach from the area of the infected mastoid to the abscess through which it was possible to work without encroaching on normal meningeal structures around it. Without doubt, it was due to this fact that the commonest of sequelae, meningitis, did not result in this instance.

Equally important, from the operative point of view, is a surgical classification of abscess of the brain, in which most authors commonly recognize three divisions: adjacent, metastatic and traumatic. The latter two are not considered here. The adjacent cases are, as the name implies, those resulting from extension of the infection from a nearby focus, in this instance the mastoid. They are further subdivided according to whether they are of long standing and hence possess a definite walling-off capsule or acute and without this protective structure. The capsule is the result of nature's effort to wall off the intracerebral infection, and is dependent on bodily resistance and the virulence of the infecting organism. According to Eagleton,[3] it requires from twenty-four to thirty-eight days before this capsule is sufficiently dense to offer appreciable resistance to a semiblunt searcher. This resistance was definitely felt at punctures in the case reported here, and there is no hesitance in classifying the abscess as chronic.

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3. Eagleton, Wells P.: Brain Abscess, New York, The Macmillan Company, 1922.

The presence or absence of a capsule has a most important bearing on the procedure to be carried out at the time of operation. If there is no evidence of capsule formation, simple puncture is advisable for the relief of pressure, and should be made through the stalk if present. One may then delay until capsule formation takes place and the adhesions about the stalk become dense and secure enough to warrant a second attempt at more permanent drainage. Evacuation at this time should, of course, be as complete as possible, and it may be best to leave a drainage tube in place during the formation of the capsule. In those cases in which it can be demonstrated that a capsule is present, evacuation should be more thorough; the abscess cavity should be inspected with some form of endoscopic instrument, and permanent collapse and obliteration secured if possible. The question of drainage is a much discussed one, various authors preferring various methods, from the simple catheter to the ingenious wire basket of Mosher. More recently King has reported excellent results from decompression and removal of enough of the cortex of the brain to allow the abscess gradually to herniate outward and become extruded from the cranial tissues.

Another important question is the best approach to the abscess, of which there are two most generally favored. The first is through the mastoid process, recommended chiefly for abscesses of the subtemporal lobe rising from an infection of the middle ear, particularly if a stalk and capsule formulation are suspected. Objections to this approach are that it traverses an infected area, that it may miss a subdural abscess, and traumatize uninvolved brain tissue, and that it fails to afford sufficient room to perform an adequate decompression. For these reasons some surgeons prefer an osteoplastic operation with exposure of a large area of brain, draining either directly or through the middle ear by enlarging this cavity outward.

In this case, the problem of operation was complicated by the initial symptoms of meningitis, the first operation being designed largely to combat this condition. When it was finally decided to explore, the right temporal lobe already had an area of exposed and presumably adherent dura; and it seemed far more logical to explore through this walled off track or stalk than through any separate and more exposed surface. The ease and promptness with which pus was recovered justified this procedure, although in another instance it might prove unwise. In the light of subsequent events, it was a mistake not to provide some form of permanent drainage at the time of the first exploration, although at that time it was felt that with almost complete evacuation pus might not form again, or that, if it did, it could be similarly evacuated without risk of meningitis. This was found to be true, but it was largely a matter of good fortune, though no difficulty was experienced in reentering the abscess cavity. This was undoubtedly due to the fact

that it was so completely distended with gas as to present an ideal surface for puncture. The formation of gas was a most unexpected and unusual development and one for which I was totally unprepared, having suspected the return of intracranial pressure to be due either to more pus or to blood.

The convalescence was remarkable for its lack of complications. There was never any suggestion of returning intracranial pressure, irrigation of the abscess cavity was easily carried out, and the drainage tube was spontaneously extruded in a short time. Since then the patient has been well, with the exception of a slight aural discharge from the right ear.

CONCLUSIONS

1. The colon bacillus is a rare etiologic factor in abscess of the brain.

2. Protective meningitis, with typical symptoms of a general meningeal involvement, may be the first sign of intracerebral abscess.

3. This type of meningitis may sometimes be relieved by removal of the focus of infection and exposure of the dura.

4. Treatment of the abscess can sometimes better be deferred until a definite diagnosis and localization can be made.

5. The best results in drainage come from evacuation of the abscess with minimum trauma to the adjacent meninges.

DEAFNESS IN SCHOOL CHILDREN

DIFFERENTIAL DIAGNOSIS WITH THE AID OF AUDIOMETERS; EXAMINATION OF ONE THOUSAND PUPILS

EDMUND PRINCE FOWLER, M.D.
NEW YORK

For the purposes of this study my co-workers and I selected a public school in the city of New York, having 1,000 pupils in excellent health and social surroundings. The children, the school and the equipment are the best we have ever seen. The neighborhood is comparatively quiet. In several other schools in which the children have been examined during the past two years, the percentages of deafness have been greater than, and in some cases double, those found in this school.

What is the best method for examining the hearing of school children? To determine this, Drs. Douglas McFarland of Philadelphia, Horace Newhart of Minneapolis, F. W. Bock of Rochester and I have coöperated with Harvey Fletcher of the Bell Telephone Laboratories, New York. After repeated tests in various rooms and under various acoustic conditions, the whispered voice changing in loudness or with distance from the pupils was abandoned for group testing by means of the phonograph-audiometer, because the results obtained otherwise were too unreliable, and the time consumed excessive. Likewise the watch-tick and tuning fork tests were found useful only in testing individuals. All other methods needed experienced testers and had the grave defect of not determining the amount of monaural hearing loss when the other ear is normal.

The phonograph-audiometer (4A) substitutes for the speech of the tester a record of the same numbers used in the actual speech tests so recorded that each group of figures may be reproduced with a difference in their intensity of 3 sensation units. This graded diminution in intensity begins 30 sensation units louder than the normal hearing capacity and descends to 3 sensation units fainter than normal. Children showing a loss of 9 or more sensation units were considered defective. A questionnaire was also submitted to all pupils, as shown in figure 1. It was found that with this method, in groups of forty, as many as 150 pupils could be tested in an hour. One hundred and fifty pupils, most of whom were under 6 years of age, were unsatisfactory subjects and were omitted from the data. Figure 1 shows a typical chart of a pupil and figure 2, the method of checking the responses with the aid of a master record sheet.

Table 1 shows the results obtained in a class of thirty-eight pupils from grade 6-B.

Table 2 gives a summary of the results obtained in the whole school, using the 4A audiometer. This table is divided into two parts: The upper part shows the number and the per cent of ears in which there

TABLE 1.—*Result of a Hearing Test Made on Pupils in Grade 6-B*

Questions Comprising the History

1. Did you ever have an ache or pain in your ear?..... Which ear?..... When?.....
2. Did your ever have a running ear?..... Which ear?..... When?.....
3. Does it run now?.....
4. Do you ever have noises in your ear, like buzzing, hissing or roaring?..... Which ear?..... When?.....

Name	Age	Date of Tests, March 16, 19— Right Ear	Left Ear	History 1	2	3	4
1. B. W.	10	—3	—3	Y,b. 5 yrs. ago*	.....	.....	Y,l. last month
2. L. H.	11	0	—3	.....	.....	.....	.....
3. B. K.	12	—3	0	Y,r. 2 yrs. ago	.....	.....	.....
4. E. G.	11	0	—3	Y,l. Dec. 1925	Y,l. Dec. 1925	.....	.....
5. E. F.	11	—3	0	.....	.....	.....	.....
6. W. P.	11	—3	0	.....	.....	.....	Y,r. last year
7. A. B.	12	0	—3	Y,r. 3 yrs. ago	.....	.....	.....
8. H. F.	11	0	0	.....	.....	.....	.....
9. C. G.	11	0	0	.....	.....	.....	.....
10. E. H.	10	0	0	.....	Y,r. 2 yrs. ago	.....	Y,r. 2 yrs. ago
11. C. S.	13	0	0	.....	.....	.....	.....
12. G. M.	11	0	0	.....	.....	.....	.....
13. G. D.	12	3	0	.....	.....	.....	.....
14. H. K.	10	3	0	.....	.....	.....	.....
15. E. H.	13	0	3	.....	Y,b. 18 mos. ago	Abscess l.	.....
16. C. L.	10	0	3	Y,r. 2 wks. ago	.....	.....	Y,r. last year
17. E. M.	10	0	3	.....	.....	.....	.....
18. H. C.	10	3	3	.....	.....	.....	.....
19. D. F.	11	3	6	Y,b. last week	.....	.....	Y,b. last week
20. O. K.	10	3	6	.....	.....	.....	.....
21. L. W.	11	3	6	Y,r. 3 wks. ago	Y,r. 3 wks. ago	.....	Y,r. 3 wks. ago
22. R. B.	10	6	3	.....	.....	.....	.....
23. R. P.	10	3	6	.....	.....	.....	.....
24. A. W.	13	3	6	Y,l. Feb. 1925	.....	.....	Y,l. occasionally
25. J. P.	10	6	3	.....	.....	.....	.....
26. D. J.	11	6	3	Y,b. 3 yrs. ago	Y,b. 3 yrs. ago	.....	Y, noise in left
27. E. J.	12	6	3	Dr. treated r. ear	.....	.....	.....
28. W. R.	11	6	3	Y,r. with recent cold	.....	.....	Y,r. lately
29. D. M.	10	3	6	Y,r. last winter	.....	.....	Y,b. last winter
30. H. D.	10	3	6	.....	.....	.....	.....
32. B. S.	11	6	3	Y,r. 2 yrs. ago	Y,b. 2 yrs. ago	.....	.....
32. M. B.	11	6	6	.....	.....	.....	.....
33. N. B.	10	6	6	Y,b. occasionally	Y,r. a year ago	.....	.....
34. H. S.	10	12	3	Y,r. 1-2 days ago	.....	.....	.....
35. E. D.	12	15	0	Y,r. week ago	Y,r. week ago	.....	.....
36. L. P.	11	15	18	Y,b. week ago	Medical treatment	.....	Y,b. often
37. W. C.	10	24	9	Y,r. at 6 yrs.	Y,r. at 6 yrs.	Mastoid	.....
38. D. S.	11	24	6	Y,l. a mo. ago	.....	.....	Y,l. a mo. ago

* Y indicates yes; l, left ear; r, right ear; b, both ears.

TABLE 2.—*Summary of Results on All Pupils in School in Which Examination Was Made*

All Ears

	−3	0	3	6	9	12	15	18	21	24	27	30+
Loss of hearing*												
Number of ears	317	518	528	237	33	12	6	7	4	2	2	6
Per cent	19.0	31.0	31.6	14.2	2.0	0.7	0.35	0.4	0.2	0.1	0.1	0.35

Best Ears

	−3	0	3	6	9	12	15	18	21	24	27	30+
Loss in hearing†												
Number of pupils	284	302	191	42	8	4	2	0	1	0	0	2
Per cent	0.34	36.1	22.8	5.0	0.96	0.48	0.24	0	0.12	0	0	0.24

* Number of pupils having a hearing loss of 9 or greater in one ear only, 38.
† Number of pupils having a hearing loss of 9 or greater in both ears, 17.

were losses of hearing corresponding to the figures at the top of each column (the loss in hearing is given in sensation units). The lower part gives similar data on only the better ear of each child. It will be seen that the number of pupils having a loss of 9 or more sensation units in one ear only was 38 and in both ears, 17, making a total number of 55. By subsequently adding 6, there were 61 who had a loss of 9 or more sensation units.

After retesting those in whom the results were doubtful, all those with a loss in hearing of 9 sensation units or less were tested by the 2A audiometer, and audiograms of both ears were made.[1] The examination was continued as follows:

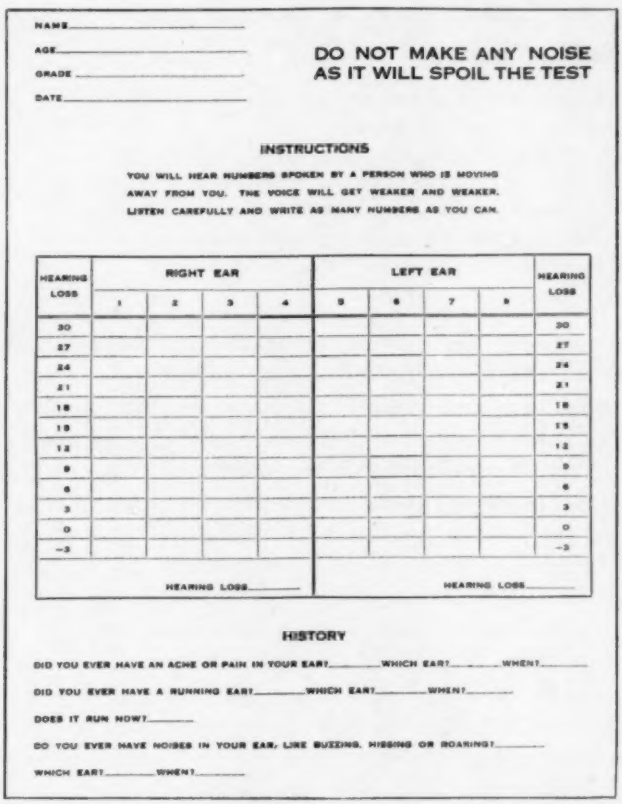
1. A check-up on the child's answers on the blank form.
2. Examination of nose and throat.
3. Inspection of ear drums, especially as to mobility, adhesions, congestions and perforations.
4. Determination of presence or absence of middle-ear negative air pressure.
5. Bone conduction (merely as to its being above or below normal).
6. Noise apparatus, if indicated.
7. Galton whistle.

About twenty persons an hour may be thus examined and a diagnosis made, with the aid of the history and audiogram. In a few persons, ten or more minutes were required to determine their lesion more accurately (three cases indicated a diminished bone conduction and four a lowered high tone limit with the Galton whistle). In arriving at a diagnosis we relied mainly on two things: the history and the audiogram. The first is often elusive, but we decided to designate those cases in which there were histories of repeated ear aches or discharges as O.M.S. (otitis media suppurativa) recurrent; those in which there were single or few ear aches or abscesses as O. M. S. healed and those in which there were persistent suppurations as O. M. S. chronic, adding C for chronic; H for healed and R for recurrent, as indicated. Those cases in which there were no history or signs of scars were diagnosed as (otitis media catarrhalis chronica). Any detectable nerve involvement was, of course, added to the diagnosis. The audiogram was indispensible, because on inspection a majority of the deafened ears appeared no different from scores of ears possessing apparently average normal hearing, and a majority of the apparently normal companion ears gave a subnormal reaction.

I do not believe that a retracted drum, a thinned drum, or a drum with lessened luster or with distorted light reflex must necessarily be associated with detectable lowered hearing. The observations in these children certainly sustain this unbelief. Furthermore, a diagnosis of negative air pressure in the middle ear, from the appearance of the drum

1. For full details of the methods and apparatus see Fowler and Fletcher, "Three Million Deafened School Children," J. A. M. A. **87**:1877 (Dec. 4) 1926.

membrane, is practically impossible. It is necessary only to make it a practice to test the patency of the tube under plus and minus Siegel air pressures and the action of swallowing to be convinced that no middle-ear air pressures remain pathologically negative for any length of time. For our diagnosis in these cases of deafness, we relied largely on the audiogram; in other words, on the relative loss in sensation units at the different frequencies. This is different from the varying number of seconds or loss in tests with tuning forks, as has been repeatedly pointed out.



NAME_____

AGE_____

GRADE _____

DATE_____

DO NOT MAKE ANY NOISE
AS IT WILL SPOIL THE TEST

INSTRUCTIONS

YOU WILL HEAR NUMBERS SPOKEN BY A PERSON WHO IS MOVING AWAY FROM YOU. THE VOICE WILL GET WEAKER AND WEAKER. LISTEN CAREFULLY AND WRITE AS MANY NUMBERS AS YOU CAN.

HEARING LOSS	RIGHT EAR				LEFT EAR				HEARING LOSS
	1	2	3	4	5	6	7	8	
30									30
27									27
24									24
21									21
18									18
15									15
12									12
9									9
6									6
3									3
0									0
−3									−3
	HEARING LOSS_____				HEARING LOSS_____				

HISTORY

DID YOU EVER HAVE AN ACHE OR PAIN IN YOUR EAR?_____ WHICH EAR?_____ WHEN?_____

DID YOU EVER HAVE A RUNNING EAR?_____ WHICH EAR?_____ WHEN?_____

DOES IT RUN NOW?_____

DO YOU EVER HAVE NOISES IN YOUR EAR, LIKE BUZZING, HISSING OR ROARING?_____

WHICH EAR?_____ WHEN?_____

Fig. 1.—A typical chart for use by the pupil.

It were better had we been able to take audiograms of the bone conduction, using the method that I proposed last year, but this was impossible because of the time required. However, bone conduction was estimated in the usual manner (with 256 and 512 double vibration forks) as being normal, or above or below normal. In suspected nerve lesions the higher forks were also employed, with noise apparatus masking the opposite ear. Can a differential diagnosis be made from the history, inspection and audiogram?

The audiograms of these cases show certain characteristics which broadly may be used to group them into three distinct types, each type having three grades. These characteristics are:

1. The direction of the slope of the curve (i. e., ascending or descending) as read from left to right.
2. Absence of any marked tendency to slope (up or down).
3. Distance of the curve below normal or below that of the opposite ear.
4. An abrupt ascent or descent near the right of any curve.

Of course a great many variations often appear, but it is well for the present to limit the types as indicated.

CLASSIFICATION OF AUDIOGRAMS ACCORDING TO TYPE AND GRADES

A1: Ascending. Loss in lower tones not over 30 sensation units; higher tones, 15 sensation units or more above lower.

A2: Ascending. Loss in lower tones more than 30 sensation units; higher tones, 15 sensation units or more above lower.

A3: Sharp ascending termination to any curve.

B1: Horizontal (or Balanced). Loss on the average of not over 30 sensation units.

B2: Horizontal (or Balanced). Loss of over 30 sensation units and not over 45 sensation units below better ear.

B3. Horizontal (or Balanced). Loss of over 45 sensation units below the better ear or the normal at middle frequencies.

C1: Descending. Lower tones 15 sensation units better than higher; loss in higher tones not over 35 sensation units.

C2: Descending. Lower tones 15 sensation units better than higher; loss in higher tones more than 35 sensation units.

C3: Sharp descending termination to any curve.

Figure 3 shows some audiograms of actual cases classified according to type and grade.

The third grade in each type may be basically but a variation of the first or second form of the same or other types, but for convenience of classification I present the following arrangement.

The type is determined principally by frequencies below 8,000. I shall discuss briefly each type of curve and the diagnoses made with its aid. The graph of the worse ear in every case determined the grouping.

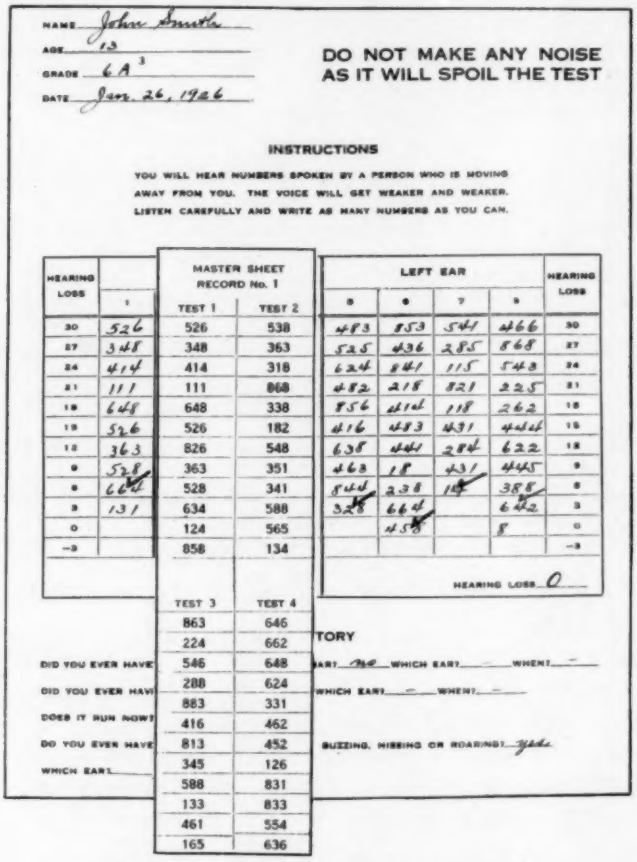
A1.—This group shows a typical picture of obstructive deafness. We have so interpreted it. Nine cases were otitis media catarrhalis chronica, and five were healed suppurations.

A2.—This type might in some cases be classed as a combined obstructive and nerve deafness on account of the lowering of the upper tones, unless bone conduction is normal; in every pupil examined it was normal. We diagnosed all cases in this class as obstructive deafness. All seven were cases of otitis media catarrhalis chronica or mastoiditis, healed.[2]

2. No tone gaps were found in any of these cases, nor in the thousands now charted from our audiometer data.

B1.—The sixteen ears in this group were not suppurating. One had increased bone conduction. We diagnosed according to past and present history. There was little loss of hearing in any case. Eight were cases of otitis media catarrhalis chronica, seven healed suppurations, and one acute.

B2.—The worst ears in eleven cases were found in pupils with a history of past or present suppurations; four were operated on for



NAME John Smith
AGE 13
GRADE 6A 3
DATE Jan. 26, 1926

DO NOT MAKE ANY NOISE AS IT WILL SPOIL THE TEST

INSTRUCTIONS

YOU WILL HEAR NUMBERS SPOKEN BY A PERSON WHO IS MOVING AWAY FROM YOU. THE VOICE WILL GET WEAKER AND WEAKER. LISTEN CAREFULLY AND WRITE AS MANY NUMBERS AS YOU CAN.

HEARING LOSS	1	MASTER SHEET RECORD No. 1 TEST 1	TEST 2	TEST 3	TEST 4	LEFT EAR 5	6	7	8	HEARING LOSS
30	526	526	538	863	646	483	853	541	466	30
27	348	348	363	224	662	525	436	285	868	27
24	414	414	318	546	648	624	841	115	548	24
21	111	111	868	288	624	482	218	821	225	21
18	648	648	338	883	331	856	414	118	262	18
15	526	526	182	416	462	416	483	431	444	15
12	363	826	548	813	452	638	441	284	622	12
9	528	363	351	345	126	463	18	431	445	9
6	664	528	341	588	831	844	238	18	388	6
3	131	634	588	133	833	328	664		642	3
0		124	565	461	554		458		8	0
−3		858	134	165	636					−3

HEARING LOSS 0

TORY

DID YOU EVER HAVE ... EAR? No WHICH EAR? ... WHEN? ...
DID YOU EVER HAVE ... WHICH EAR? ... WHEN? ...
DOES IT RUN NOW? ...
DO YOU EVER HAVE ... BUZZING, HISSING OR ROARING? yes
WHICH EAR? ...

Fig. 2.—Method of checking responses.

mastoiditis. Bone conduction was normal in all. Two cases were diagnosed otitis media suppurativa chronica, healed; four otitis media suppurativa, healed, and one otitis media suppurativa, recurrent. The condition in the better ear in five cases was otitis media catarrhalis chronica; that in the better ear in six cases was otitis media suppurativa chronica, recurrent or healed (several ears classified in types B1 and B2 might also be put in A1 and A2, respectively, but the higher the grade the greater the tendency to follow the horizontal type).

Is a marked deafness equally distributed throughout the frequency range possible without nerve involvement? It is conceivable that a partial nerve deafness (affecting the lower frequencies but little and yet cutting down the higher frequencies progressively) in combination with an obstructive lesion operating in exactly the reverse order (i. e., effecting the higher frequencies but little and yet cutting down the lower frequencies progressively) might level up the curve slopes of both nerve and obstructive lesions so that the combined effect would appear approximately horizontal, or balanced. Unfortunately, no such simple

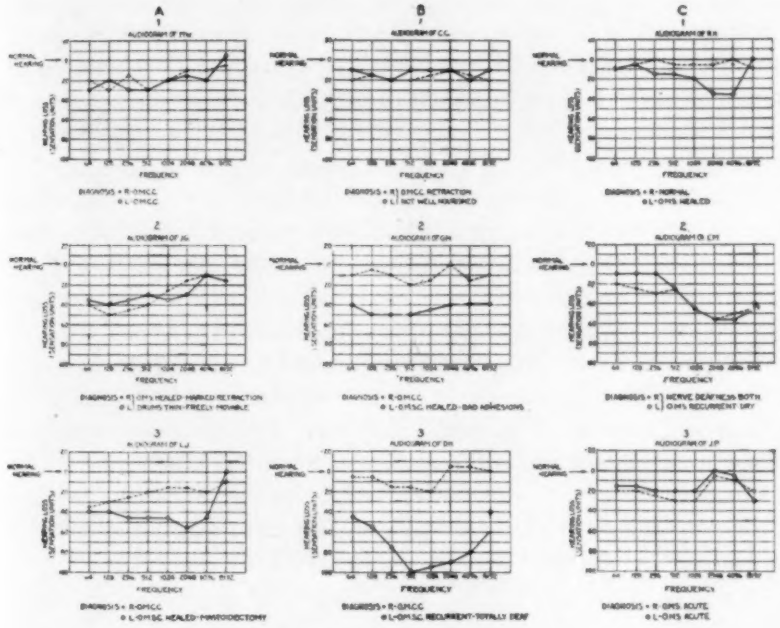


Fig. 3.—Types of audiogram curves.

solution of the problem appeals to us, as both nerve and obstructive lesions may effect both high and low frequencies, depending on the location and nature of the lesion. From the history in these cases, it is certain that there is an obstructive lesion; so it is necessary to prove that such a lesion alone may cut down the higher frequencies to the extent shown in the audiograms, if a diagnosis of nerve deafness is to be avoided. All these cases registered a loss at the higher frequencies of from 30 to 40 sensation units. Can an obstructive lesion cause such a loss at the high frequencies?

C1.—Seven cases are included in this group. All but one pupil gave histories of suppuration. The low tones were well heard. One child

showed the worse ear in this class and the better ear in class A1. The history concerning suppuration was the same for both ears. Can it be that in this case the better ear has obstructive deafness and the worse ear a combined obstructive and nerve involvement? This is possible, but the worse ear, which is below normal at the high frequencies, could suffer diminution in acuity wholly from a loading of certain parts of the conducting mechanism diminishing mainly the higher frequencies and not affecting the lower ones to any extent. The picture is obscured by such a possible state of affairs. So far as we know, attention has not been called to a loss in certain high frequencies by loading limited areas of the conductive mechanism. We here make the point that it is not only theoretically conceivable, but also practically demonstrable in certain cases in which drum tension, mobility and weight are not sufficiently abnormal to have an opposite effect, the articulations of the ossicles not relaxed and the ossicles well loaded, as by congestion or calcium deposits (to mention two).

Of course there must be a limit beyond which, in vivo, it is impossible or improbable that any loading process will lower the high tones. We have tentatively placed this limit at more than 35 (or 40) sensation units below normal.

C2.—In this group (three cases) the high notes are below 35 sensation units or not heard at the intensities available. This does not mean that they could not be heard if a greater intensity could be produced. We diagnosed these cases as nerve deafness. Two pupils gave definite histories of severe trauma, probably fractures of the skull. The results in companion ears were within the average normal limits. The third had otitis media suppurativa recurrent in both ears, which had followed pertussis and measles, and both his ears gave identical curves.

A3.—Near the right of the horizontal and descending curves a sharp ascent occurs in a considerable number of audiograms. This ascent confirms our belief that such graphs mean no involvement of the nerve or labyrinth in spite of a decided lowering of the very low middle and near high tones. The occurrence of this terminal rise is one of the reasons why we did not diagnose some of the cases, especially in class A3, as nerve deafness. A stiffening of the whole or of part of the middle ear apparatus without loading could account for such an ascent at the right of the curve.

B3.—The cases in this group are frankly cases of nerve deafness. No obstructive lesion can cause so much loss of hearing.

C3.—Audiograms frequently show a sharp descent in their curves at the higher frequencies, irrespective of the lower tone slopes. What interpretation should be placed on such terminal sharp declines? On

physical grounds they may be ascribed purely to weighting without increased ossicular point friction or interference. We have, therefore, not diagnosed these ears as having nerve deafness, no matter how low we have found their lower and middle tones, so long as the terminal drops were not below a loss of 35 sensation units. This is a more or less arbitrary decision, but there appear to be good reasons for making it. One is that bone conduction was normal in all, and we have scores of similar adult patients whose bone conduction at all frequencies was normal; another is that the ears under type B2 all show a loss of about 35 sensation units for all frequencies, with bone conduction normal. It may be that the position of the receiver over the ear partly accounts for some of these losses at high frequencies.

Physical changes may cause any of the variations of audiogram curves shown in these graphs, with the exception of those diagnosed nerve deafness. We shall discuss this at some future time.

Table 3 shows the number of cases in each class, the diagnoses in both ears and the number of cases with similar curves in companion ears, also the diagnoses in better ears.

We have compared the graphs of companion ears as to form, because we believe that similarity in form means similarity in the physical or acoustic conditions of the two ears (but not necessarily vice versa), hence also a probability of similar lesions, though a history of such lesions may be unobtainable. It is impossible to conceive of the hearing capacity in two ears varying equally and depending on frequency, unless such a coincidence is largely dependent on acoustic characteristics common to both.

In forty cases (two thirds of the group) both ears showed similar lesions, the diagnoses differing with the history. Omitting six normal better ears (two better ears with total deafness in the worse ears), in all of which a great dissimilarity of lesions and curves is to be expected, there remain but thirteen pupils (21-8) with markedly differing disease processes in the two ears.

In forty-nine cases (five-sixths of the group) the graphs of both ears were similar in form even if not in type (this is commonly found also in adult ears). Omitting the three cases of total deafness in B3 and the two pupils with normal better ears in C2, there remain but seven cases (12-5) in which the graphs of both ears were different in form.

In subtypes A3, B3 and C3 there are greater difference between the worse and the better ears in similarity of audiograph form, but only eight of the twenty-seven in these classes differ in similarity of lesion. The reason this is so in A3 and C3 is that these subclasses are made up wholly of the higher grades from all types; the reason in B3 is that there is total deafness in the worse ears.

TABLE 3.—*Diagnoses in 61 Cases of Deafness in Children Between the Ages of 6 and 13 Years, Arranged Under Types According to the Audiograms of Their Worse Ears*

TYPE AND GRADE	WORSE EAR	BETTER EAR	TOTALS	BOTH EARS SIMILAR IN LESION	BOTH EARS SIMILAR IN CURVE FORM
A_1	CMCC 9 CMSCH 2 CMSH 2 CMSR 1	9 2 1 CMCC 1 1	14	13	14
A_2	CMSH 5 MAST.N 2	3 CMCC 2 CMSH 1 CMSC 1	7	5	7
		TOTALS	21	18	21
A_3 Included in other types.	CMCC 5 MAST.N 4 CMSR 2 CMS Ac 1 CMSH 2	5 CMCC 3 CMSH 1 H 2 H 1 H 1 NORMAL 1	14	10	6
B_1	CMCC 8 CMSH 4 MAST. 2 CMSCH 1 CMS Ac 1	CMSH 7 CMSH 1 NORMAL 1 CMCC 2 CMCC 1 Ac 1	16	11	15
B_2	MAST.N 4 CMSH 4 CMSCH 2 CMSR 1	CMSH 1 CMCC 3 CMSH 2 CMCC 1 NORMAL 1 CMSH 1 CMCC 1	11	5	10
B_3	MAST.N 1 CMSR 1 CMSH 1 (All total deafness)	CMCC 1 CMCC 1 NORMAL 1	3	0	0
		TOTALS	30	16	25
C_1	CMSR 3 CMCC 1 CMS Ac 1 MAST.N 1 CMSH 1	3 1 H 1 CMSC 1 NORMAL 1	7	5	2*
C_2	NERVE 3	1 NORMAL 2	3	1	1
		TOTALS	10	6	3
C_3 Included in other types.	CMCC 4 CMSCH 4 CMSR 1 CMSH 1	4 4 1 CMCC 1	10	9	6

*Difference was at higher frequencies only.

DIAGNOSES IN WORSE EARS

CMCC	18	17 in Type A_1, and B_1, 1 in C_1 (5 in Sub Class A_3, 4 in Sub Class C_3, None in Class A_2, B_2, C_2 or B_3)
CMS	37	5 CMSCH 2 in Type A, 6 CMSR 1 in Type A, 16 CMSH 7 in Type A, 2 CMS Ac 0 in Type A, 9 MAST. 2 in Type A 3 " " B, 1 " " B, 6 " " B, 1 " " B, 6 " " B 0 " " C, 3 " " C, 1 " " C, 1 " " C, 1 " " C
Nerve	6	(Total Deafness - 3, in Type B_3, all with history of chronic suppuration) (Partial Deafness - 3, in Type C_2, one with history of chronic suppuration two with history of probable fracture of skull)
TOTAL	61	Only 14 of whom, (1/4), admitted any defect in hearing in either ear. 90% show binaural disease.

DIAGNOSES IN BETTER EARS

CMCC	31
CMS (A.C.H. or H.)	23
Nerve Deafness	1
Normal	6
TOTAL	61

The relative coincidence of a history of disease of the ears with the determined deafness was as follows:

Number of pupils with a negative history and with a loss of less than 9 sensation units in hearing	414
Number of pupils with a positive history and with a loss of less than 9 sensation units in hearing	361
Number of pupils with a positive history and with a loss of 9 or more sensation units in hearing	44
Number of pupils with a negative history and with a loss of 9 or more sensation units in hearing	17
Number not examined	155
	991

The percentage of deafness found in the pupils examined was 7.3.

It is certain that nearly all of the pupils with a positive history of ear trouble and many with negative histories have not normal ears, although their hearing is now little affected. Of 361 pupils with hearing better than 9 sensation units there were 240 with histories of ear aches and running ears, and 71 with tinnitus. How many and which of these children will subsequently suffer from deafness? (How many of the 414 without positive histories of ear trouble will subsequently suffer in this manner?) How many of the 61 with definitely impaired hearing will go on to greater deafness? Regular yearly examinations would tell us something about these questions and also whether there are any characteristic audiogram types, grades or forms which presage otosclerosis or progressive, obstructive and nerve deafness. I believe that the standard of a loss of 9 sensation units chosen in these cases to denote defective hearing is too conservative. This is equivalent to two fifths of the average maximum hearing distance for speech. Had we taken a loss of 6 sensation units instead of 9 sensation units, the percentage of defective ears would have been at least doubled. Our retests indicate this, but subsequent testing will better determine it. The data herein presented are interesting and true. The practical value is obvious in some respects, and not yet determined in others.

DISCUSSION

MR. R. L. WEGEL: Dr. Fowler's paper is of considerable interest to me from the standpoint of the mechanical analysis he has made of his diagnosis. With the help of the audiogram and other more or less concise data obtained by simple observation, he has suggested tentative hypotheses regarding the specific seat or seats of a lesion.

The general trend of effort in interpreting audiogram curves has apparently been to correlate empirically their shapes and levels with pathologic conditions as described in terms of the current otologic classification. This work has apparently met with sufficient success to show considerable possibilities, although the correlation has not been made as definite as might be desired. The question naturally arises—why has it not been possible to diagnose with greater certainty by means of the audiometer supplemented by such other tests as are in

common use? This may be due to a number of causes, such as a lack of sufficient knowledge to make interpretations with the desirable degree of perfection, inadequacy of the measurements and faults in the currently used classification of the various pathologic conditions.

It would seem that a profitable procedure for the reduction of these uncertainties would be to make a more careful study of the mechanics of normal and pathologic hearing. To begin with, a working hypothesis must be adopted, and for this purpose the so-called "Helmholtz Theory" offers the most promise.

If a thin piece of paper about 5 inches wide was pasted across the bank of strings on a piano, and then if black and white keys were painted over the strings so as to give the appearance of a keyboard, the painted keys would vibrate as the corresponding keys of the real keyboard were struck. If a piece of music was played, the appearance of the painted keyboard would be much the same as that of the real keyboard except that the keys of the former would be blurred instead of depressed, owing to their vibration. The motion of the bass strings would be easily visible to the naked eye, whereas the higher pitched ones would have to be viewed with a microscope. If another piano in the same room was played, the painted keyboard would present the same appearance as before, because the sound board picks up vibration from the air and transmits them to the strings, causing them to vibrate in much the same way as though struck by their own associated keys. Amplitudes of motion would of course be much smaller, and a high power microscope would have to be used for one to see the vibrations in this case.

Suppose it were possible to paste a lightweight photomicrograph (about 30 mm. in length) of the piano sounding-board on the basilar membrane of one of the ears of a subject and to view this with a high power microscope while the piano was being played. The keyboard on the basilar membrane would perform much like the painted one on the piano strings. In the modern piano there are twelve notes to the octave and usually three strings to each note in the treble range, making thirty-six strings to the octave. According to Retzius, there are about 24,000 fibers in the basilar membrane, or about seven hundred per millimeter. Each octave on the basilar membrane covers about 5 mm., so that it includes about 3,500 fibers or strings. Since there are so many fibers in the interval of a half tone, a large number would respond to each note. The basilar membrane can therefore respond to tones between the piano keys with much greater facilities than the strings of a piano. Since the highest note of a piano is a little over 4,000 double vibrations, the keyboard on the basilar membrane would fail to cover the membrane completely by about 5 or 6 mm. on the proximal end.

The ears of a normal subject are presumably nearly alike in their operation. The ears of different normal subjects must vary somewhat in their operation. They all have the characteristic in common, however, that the sensitivity or acuity is greatest in the range of from 1,000 to 5,000 double vibrations and are comparatively insensitive for lower and for higher frequencies than these. If, while viewing the keyboard on the basilar membrane, one presented to the ear a tone of continuously varying pitch, from say 16 double vibrations to 16,000 double vibrations, a small amplitude of motion covering a rather wide area near the helicotrema would be observed at the start, and as the pitch increased (the sound intensity of course being kept the same), the vibrating area would contract somewhat and move toward the round window and increase considerably in amplitude of motion. This increase would continue until the pitch was about 1,000 double vibrations, at which time the area would be close to the middle of the membrane. If the pitch is increased above, the vibrating area would remain about constant in amplitude as it moved up to a point 5 mm. from the round window, at which time the pitch would be about 4,000 or 5,000 double vibrations. As the pitch continued to increase, the intensity of the sound presumed to be constant of course, the vibrating area would move slowly toward the proximal end of the basilar membrane, decreasing somewhat in width and decreasing greatly in amplitude.

Let us consider a simple hypothetic lesion in which, over a length interval of about 5 mm. near the center of the basilar membrane, the tension in the fibers was doubled. If the same experiment were performed with the continuously varying tone, when it reached a pitch of 1,000 cycles it would be observed that the vibrating area, instead of being at the middle of the membrane as before, would be displaced about 2.5 mm. closer to the round window. In approaching 1,000 double vibrations, the travel along the basilar membrane of the vibrating or blurred area would be much accelerated and in leaving, it would be slow. At other pitches than those of the mid-range the performance of the ear mechanism would be sensibly the same as before the lesion occurred. The amplitude at any time, however, would in either case be about the same throughout the entire range.

The audiogram might be considered as a curve showing the reciprocal of the amplitude of the motion of the basilar membrane as measured with the microscope in this experiment. As the amplitudes before and after this "lesion" had taken effect would be about the same, the audiograms in the two cases would be expected not to differ greatly. There would, of course, be differences, but these would probably not be much greater than those observed ordinarily in normal ears. The patient, however, would probably lose, to a certain extent, his ability to enjoy music. His faculty of telling the direction from which a sound comes (binaural sense) would perhaps be impaired. If his other ear was normal, he would complain of diplacusis.

The point which I have tried to make by illustration of a hypothetic lesion, regardless of the physical possibility of its occurrence, is that there are probably some of the finer distinctions in pathology which it is impracticable to try to investigate with the unsupplemented audiometer. Routine tests, of course, cannot involve elaborate testing methods, but from the standpoint of a research, the object of which is to investigate accurately the causes or seats of lesions, interpretations should be made in terms of a mechanical operation of the ear. Dr. Fowler is taking steps in this direction.

D. G. E. SHAMBAUGH, Chicago: The proposition brought forward in the paper by Dr. Fowler for the examination of school children is one of which we all can approve. One of the difficulties lies in the fact that it is rather a time consuming proposition. If the apparatus the Western Electric has devised will shorten materially the time of examination and give the desired data in making such examination, this objection will be partly removed. The first problem is to determine whether there is some defect in hearing. Perhaps an apparatus of this sort will detect this in groups more quickly than voice tests with individuals. The next step is to determine the cause of the deafness. This requires an otoscopic examination and such functional testing as will assist in locating the source of the defect. I have the feeling that the false note in this discussion has been struck in that too much emphasis has been placed on the point that if these examinations are regularly performed on young children by detecting the onset of the deafness in its earliest stage, much can be done toward preventing that type of chronic progressive deafness which in adults develops into a serious handicap. This is not a fair statement of the proposition. Diseases of the ear in children are of several types; the first is congenital deafness. This, of course, we cannot correct, improve or retard. Then there is nerve degeneration, toxic in origin and dependent on infections. Detection of this does not give us any clue that will assist in treatment. The treatment of the suppurative diseases of the middle ear does not influence the defect in the hearing to any great extent, a fact of which we are all aware. Then comes the tubotympanic processes, most common of all ear troubles in children. It is a common error to suppose that these processes so common in childhood are responsible for those cases of progressive deafness which in middle life produce a serious handicap. The tubotympanic disease of childhood is rarely prolonged as an active process into middle life. The chronic progressive deafness of adults may, of course, develop in a person in whose childhood there has been some tubotympanic process, but this does not

mean that it is dependent on this for its cause. Dr. Fowler discusses the defect at the upper end of the tone range. This I have always been inclined to interpret as evidence of a nerve involvement. It often occurs in connection with acute otitis media and disappears as the ear recovers from the inflammation as the defect at the lower end of the tone scale disappears. I have been inclined to look on this defect in the upper tone ranges as toxic. Dr. Fowler would attribute it to weighting down of the conducting mechanism.

Dr. Horace Newhart, Minneapolis: The simple preliminary examination of 5,000 school children with the audiometer is a relatively easy task compared with the painstaking work of reexamining, making an audiometric test and carrying out an otologic examination on the many persons in this group, and tabulating carefully the results, as Dr. Fowler has done in the preparation of his valuable contribution. This work, I believe, is of great importance in the further development of otology, for it opens large possibilities of research and therapy in our field.

The methods we are now employing in audiometry are the best and most practicable yet attained, but they are not beyond criticism and doubtless will be improved. One point to which I would call attention is the fact that as yet we have no absolute or fixed standard as to what constitutes normal hearing acuity. In audiometry we now have the means for determining this basic standard for future scientific work, and it is to be hoped that this fundamental problem will soon be worked out in a manner acceptable to otologists, physiologists and psychologists. As regards the particular problem of the school child, I have taken a slightly different attitude from that taken by Doctors Fowler and Fletcher. In their practical work in detecting the loss of hearing among school children in New York, they regard as worthy of serious consideration and further study only those with a loss of hearing of 9 or more sensation units in the better ear. In my experience, based on a preliminary survey of 5,000 persons, I feel strongly that as a preventive measure, in justice to the child, all school children who on a retest show a loss of hearing of 6 sensation units or more should be given a careful otologic examination, with a view to eliminating all possible physical causes of a further loss.

Dr. M. A. Goldstein, St. Louis: In almost all new work undertaken, we are bound to be ultrascientific. Physicists thrust on us such terms as "sensation units," "dynes" and other factors. We do not know much about them, and for the practicing otologist we can boil things down to a few practical conclusions. Take the ordinary cases with curves representing normal hearing—there are certain definite positions in the audiograms that lead to definite conclusions. We know that when we have depression of the lower tone limit and a normal upper tone limit, and when we have a variation in the intensity, we are dealing with conduction deafness. We also know that when there is a depression in the upper tone scale, no matter what the other phase of the scale may be (it need not be normal), a person has nerve deafness. If there is depression of the lower scale and upper scale (and even absence of 64 double vibrations), the physician is dealing with otosclerosis.

Dr. Harris P. Mosher, Boston: The members of the society will agree that Dr. Shambaugh stands almost alone in attainment, and he stands alone when he says that chronic progressive deafness is a condition of adult life. I think chronic progressive deafness is a condition that progresses from youth to adult life.

Dr. Edward B. Dench, New York: I have had rather peculiar experience with the audiometer. I have gone to the office of the company, and the audiogram could not be made, because the instrument broke down. I had one put in St. Luke's Hospital and because the patients were not sufficiently intelligent to tell exactly what they heard, it was a failure there. A representative from the firm brought it to my office, and the instrument broke down at the first test. I wonder how accurate these charts will be when made by physicians or nurses if the instrument breaks down in the hands of the firm that sells it.

MR. C. C. BUNCH, University of Iowa: I have been more or less interested in the interpretation of audiograms, as shown by the Western Electric audiometer and the Pitch Range audiometer which has been developed at the University of Iowa. The curves which have been shown by Dr. Fowler are not made from records with the no. 4A phonographic audiometer of the Western Electric Company, but from the records of another audiometer which tests the acuity of hearing for eight discrete tones at intervals of an octave. We have repeatedly pointed out in studies which have been presented in the otologic journals that unless all tones are tested, much information concerning the tonal range will not be obtained. In a recent number of the ARCHIVES OF OTOLARYNGOLOGY, special attention was called to this point. For example, between any two tones of the records shown from the audiometer giving eight tones there is a complete octave. There may be a complete or partial gap which must have some clinical significance; what this clinical significance may be, must be solved later. It is difficult to understand how a narrow defect covering only a few tones which require 3,000 or 4,000 times the intensity of adjoining tones can be caused by a defect in the conducting mechanism. Perhaps the microscope may give considerable information.

In the study of otosclerosis, at least three different types of records have appeared. The type shown by Dr. Goldstein has appeared during examinations, but it does not appear to be the only one. In some, tonal gaps may appear at almost any level; others show a gradual loss of acuity throughout the entire range, and a third type gives a record similar to that shown in a pure nerve lesion.

DR. EDMUND P. FOWLER: This work is being done for persons who are hard of hearing, under the auspices of the American Association of Plagues. I do not say that profound deafness always results from early ear troubles, but if these children are examined from year to year, it will be learned how much of the deafness is caused by early diseases. I did not diagnose a single case as otosclerosis. This is remarkable in any table of diagnosis of deafness which is made on a large scale. When the charts were arranged with the companion ears taken as types, it was remarkable to find how many cases of suppurative processes there had been. I think that these cases of deafness resulted from prior suppuration. No other inference appears plausible. It is easy to diagnose toxic deafness or otosclerosis, but difficult to prove them present. If a man has dead teeth, and is deaf, we call the deafness toxic; we say deafness is syphilitic if he has a four plus Wassermann reaction, but neither is necessarily true. We must make repeated accurate tests and observations to be convinced of the origin of nerve deafness. It is reasonable to infer, from the physical standpoint, that when the curves are similar, they are related to similar conditions of the two ears.

As to the number of sensation units used to denote faulty hearing, I think 6 would have been better; 9 is the number used conservatively. As to the interpretation of low tones or high, we have been taught to interpret loss of low tones as obstructive deafness and of high tones as nerve deafness, but this does not always coincide with what I find to be the case experimentally. A person may diminish high tones also by obstructural means; for instance, stop up the ears with the fingers pressed tightly on the troagdi, and note that the Galton whistle is heard not well or not at all. This, however, is not nerve deafness. If persons have normal bone conduction, they have not nerve deafness. Perhaps, theoretically the bone conduction may be increased sufficiently to overcome the defect from nerve deafness, but this does not seem plausible, at least in 99 per cent of cases, because bone conduction cannot be increased sufficiently.

In regard to intelligence and the audiometer tests, how about intelligence with tuning forks? The audiometer saves time and effort. I always also use tuning fork tests in my office. Please note that I did not say any particular curve meant a definite lesion. I avoided that statement advisedly at this time.

114 East Fifty-Fourth Street.

Clinical Notes

GRADENIGO'S SYNDROME

RICHMOND MCKINNEY, M.D., MEMPHIS, TENN.

The study of Gradenigo's syndrome presented by W. H. Sears, of Huntingdon, Pa., to the American Laryngological, Rhinological and Otological Society in 1925, as a candidate's thesis, has aroused new interest in the reporting of such cases. I have recently had a case in which this syndrome was present that was of more than passing interest to me.

History.—On Dec. 14, 1926, Edwin B., white, aged 7, was brought into the clinic with a purulent discharge from the ear, of more than one month's duration. The child appeared fairly well developed and well nourished, and had been up and about since the otitis began. The ear was cleansed with solution of silver nitrate. The faucial tonsils were unusually large, and it was learned that the patient had had previous attacks of tonsillitis. The child had marked left convergent strabismus. His mother was told to bring him back the next day for treatment of his ear.

When he was examined the next day, tonsillectomy and adenoidectomy were advised, with the hope that this might aid in clearing up the condition of the ear.

Operation and Course.—On December 17, tonsillectomy and adenoidectomy were performed, and the child made a normal recovery. While he was in the hospital his mother asked whether removal of his tonsils and adenoids would straighten the eyes. This question led to more careful investigation of the child's history, and also to the consultation of an ophthalmologist. The mother said that the left eye became crossed about two or three weeks previous to admission; a pain also developed over the left side of the face, and extended over the parietal region of the skull. Since the development of the condition of the eye, little change had been noticed in the appearance of the eye or in the character of the pain. The boy was sent to the eye clinic on December 20, where the following report was made: "Vision, both eyes, 20/20; complete paralysis of left external rectus; both outlines a little blurred. The child apparently cerebrates slowly."

On the same day a roentgenogram was made, and the following report was given: "The right mastoid is apparently normal: the left cells are almost entirely covered by fine haze, through which the trabeculae are discernible. The outline of the mastoid process is well defined." Laboratory examination revealed: white blood cells, 12,000; small lymphocytes, 49; large mononuclears, 1; megaloblasts, 1; polymorphonuclears, 44.

A turning test was made, but revealed nothing abnormal.

Second Operation.—On December 31, a mastoidectomy was done on the left side with exenteration of the entire mastoid process to the tip. All of the cells, from the root of the zygoma to the tip, were necrotic and full of granulations and pus. No dura or sinus was exposed. The diseased area extended backward toward the apex of the petrous portion of the temporal bone, in the region of the semicircular canals.

The posterior canal wall was found so necrotic that it was decided to remove this and to perform a modified radical operation.

On the afternoon of the day of operation it was noticed that the eye appeared perfectly straight, which was attributed to either the anesthetic or the relief from edema following the operation.

The child was in the hospital five days. On the second day, the outside dressing was changed, but the packing was not disturbed. On the fourth day, the iodoform packing and the stitches were removed. On January 4, the patient was discharged to the outpatient department.

The mastoid incision healed satisfactorily, and the external auditory canal was kept open sufficiently by means of packing. The pain of which the patient complained has entirely disappeared. The eye has improved markedly, and the report from the eye clinic on January 23, was as follows: "Ocular movements apparently normal. Diplopia with a red glass corrected by a four prism. The canal is now dry." On February 8, at the time of this report, the eye appeared to be normal.

NEW ANTRUM TROCAR AND ANTRUM RASP

FLETCHER D. WOODWARD, M.D., UNIVERSITY, VA.

The instrument illustrated here has been found useful in making a large opening into the maxillary sinus in the shortest possible time. It cannot be used in every case, but when there is sufficient room for its introduction or when sufficient room can be obtained by lifting the inferior turbinate, it is introduced well posteriorly through the antral wall, cutting by a sawing motion to the anterior limits. At this point its hooklike beak brings the excised piece of wall out between the two blades into the nasal cavity on withdrawal.

The time saved and the facility with which the procedure can be carried out will be appreciated both by the patient and by the physician, while at the same time an opening sufficiently large for drainage and future irrigations will be obtained with a minimum of trauma.

The instrument is being manufactured through the courtesy and cooperation of The George P. Pilling & Son Company of Philadelphia.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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OTOLOGIC AND NASAL DISEASES IN RELATION TO INTRACRANIAL DISTURBANCES

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

OTITIC BRAIN ABSCESS IN GENERAL

Lund,[1] in an article entitled "Otogenic Brain Abscess," reviews the material from the Copenhagen Hospital, which comprises fifty-six cases of otogenic brain abscess. He says that males are affected more than twice as often as females; children rarely under 1 year of age. Fifty brain abscesses originated from chronic middle ear suppuration; six were due to acute ear suppuration. Right-sided cerebral abscesses are more common than left-sided. Cerebellar abscess was observed with about equal frequency on the right and left sides. The streptococcus, which predominates in epidural perisinuous abscesses, occurs relatively less often than the pneumococcus in otogenic meningitis. In brain abscess the pneumococcus and *Bacillus coli* predominate, the latter greatly aggravating the prognosis. The fifteen cases in which *B. coli* was demonstrated terminated fatally. As distinguished from pneumococcus abscesses (with their relative tendency to membrane formation), an abscess from *B. coli* infection is usually without a membrane and is associated with considerable tissue necrosis.

In brain abscess, manifestations of meningitis are much less marked than focal and encephalitic symptoms. The majority of brain abscesses show a polybacillary infection, while meningitis is monobacterial. The cerebrospinal fluid was sterile in most cases of abscess; in eight cases microbes were found, with polybacillary growth in five. This may prove of value in differential diagnosis, as polybacillary cerebrospinal fluid points to a secondary meningitis; that is, another intracranial complication, generally a brain abscess, is present. The demonstration of the coli bacillus likewise points to abscess.

Cerebellar nystagmus is explained by the author as due to action on the Deiters' nuclei, probably referable to propagation of the inflammation to the vestibular nuclei in the medulla oblongata from collateral

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1. Lund, R.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:34, 1926.

edema, or actual inflammation (encephalitis). In ten cases cerebellar nystagmus was at first unilateral; in seven it was directed toward the affected side, and in three toward the healthy side. In two cases, nystagmus in the opposite direction was noted at a later date. The author assumes that in the cases in which the nystagmus is exclusively to the affected side, it is referable to irritation of the vestibular nucleus. When, on the contrary, the nystagmus is to the healthy side, it may be assumed that Deiters' nuclei are the site of a pronounced inflammatory process. Hence, an altered direction of the nystagmus indicates an extension, and those cases in which the nystagmus is to the healthy side from the beginning, should have a worse prognosis. This was confirmed by the author's experience. In one of his cases (preceding otolith disturbances in the affected labyrinth) the cerebellar nystagmus was modified by the attitude of the head.

Reliable information concerning the cerebrospinal fluid, is obtainable only through a series of punctures. There is practically always a cellular increase, the leukocytes being rather more common than the lymphocytes. The cerebrospinal fluid often clears during the formation of a brain abscess, sometimes with persistence or actual increase of the intracranial symptoms. Bradycardia of vestibular origin is relatively common in brain abscess and in otogenic acute encephalitis. A unilaterally acting localized pressure may be the most essential, although not the only, cause of bradycardia. In the cases of brain abscess in which choked disk was demonstrated, pronounced bradycardia was always noted. Among sixty-five cases in which there was slow pulse (otogenic meningitis, sepsis, brain abscess, encephalitis and subdural abscess) a mortality of 81 per cent occurred, while the mortality among eighty-six cases with a pulse corresponding to the temperature was only 32 per cent. The chief cause of slow pulse seems to be toxic irritation of the vagus. Bradycardia is a bad augury and can probably be likened to the bradycardia which is sometimes observed at the height of acute infectious diseases.

CEREBRAL ABSCESS SECONDARY TO LONGITUDINAL SINUS THROMBOSIS

On the basis of a cured case of otitic longitudinal sinus thrombosis, with sequestration of the skull bone, meningitis and brain abscess, Hirsch [2] shows that something can be accomplished by purposeful procedures when the infection does not take an over-violent course and when the natural defensive forces of the body remain intact. The patient was operated on six times. The brain abscess was discovered

2. Hirsch, M.: Diagnostic and Therapeutic Comments on a Cured Case of Longitudinal Sinus Thrombosis and Temporal Lobe Abscess, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **116**:31, 1926.

only after repeated puncture, at intervals. When the general manifestations persist after the sinus has been opened, the eventual advance of the thrombosis into the cerebral tissue must always be kept in mind.

Symptoms of Brain Abscess.—Goldflam,[3] in a "Contribution to the Symptomatology of Temporal Lobe Abscess," noted as an early symptom continuous pains in the homolateral eye, more rarely in the entire area of the first trigeminal branch. The pain is evidently dependent on the collection of pus and disappears on evacuation of the abscess. It is referred to a compression of the first trigeminal branch through the swollen temporal lobe, but it remains an open question why often only the sensory fibers to the eyeball are involved. With respect to the differential diagnosis, the author describes a case in which a patient who suffered from chronic otitis media had a meningeal hemorrhage as a result of traumatism, the bloody extravasation damaging primarily the temporal lobe. The diagnosis of hemorrhage was based on the demonstration of blood in the cerebrospinal fluid and the immediate loss of consciousness. In the differentiation of tumor from abscess, roentgenographic signs of pressure on the brain are important, especially changes of the sella turcica. Choked disk was noted by the author in 54 per cent of the cases of abscess. The simultaneous occurrence of purulent otitis media and epidemic meningitis is illustrated by a case observed in which the patient finally died of cerebellar abscess, but in which bacteriologic confirmation was lacking. In a case of left-sided temporal lobe abscess, attacks of aural tinnitus were observed in the right ear, with motor aphasia. The occurrence of aphasia in a right-sided temporal lobe abscess in a right-handed person, he regards as due to a referred pressure on the left temporal lobe.

(*a*) Aphasia in Abscess: Lund[4] states that amnesic amnesia and paraphasia are the most common and most easily demonstrable forms of disturbances of speech in temporal lobe abscesses. Pronounced word deafness was found in only a small number of cases. Verbal amnesia may produce an apparent motor aphasia. This is probably the reason that the reports in the literature state that a motor aphasia exists in about one half of the cases of a left-sided temporal lobe abscess with aphasic disturbances. Undoubtedly, a pure motor aphasia is occasionally observed (in one out of six of the author's cases with an aphasic disturbance). The motor aphasia is probably referable to encephalitis surrounding the abscess. The degree of the aphasic disturbances is not proportionate to the size of the abscess but rather to the extent of the surrounding encephalitis. Five cases of left-sided

3. Goldflam, I.: Deutsche Ztschr. f. Nervenh. **90**:38, 1926.

4. Lund, R.: Comments on Aphasia in Otogenic Abscess in the Temporal Lobe, Hospitalistid. **69**:777, 1926.

temporal lobe abscess showed no aphasic disturbances at any time in the course of the disease. Absence of aphasia, therefore, need not be a contraindication to an operative intervention. The onset of contralateral hardness of hearing possesses no diagnostic value.

(*b*) Hemiplegia: That a complete contralateral hemiplegia may be associated with extradural suppuration is shown by a case reported by Martin [5] in a child, aged 5. The operation revealed an extensive extradural abscess in the middle cranial fossa and was followed by recovery, with complete subsidence of the paralytic phenomena. That subdural abscess may clinically simulate an intracerebellar or intracerebral suppuration is shown by Mayer's [6] case of "subdural abscess at the convexity of the cerebrum after acute middle ear suppuration." The bone was found to be extensively destroyed. The dura was covered with a greenish-white membrane, which extended to the tip of the pyramid. The sinus was thrombotic. Search of the temporal lobe and cerebellum proved negative. On the fifth day after the operation meningitis supervened, and death occurred on the fifty-second day. Autopsy revealed a subdural abscess which extended over the convexity of the temporal, parietal and frontal lobes without macroscopically demonstrable connection with the primary focus. The nystagmus toward the diseased side (which had induced the author to assume a cerebellar abscess) was produced, in his opinion, by pressure on the posterior cranial fossa. He recommends, when results of the puncture of the temporal lobe and cerebellum are negative, looking for subdural suppuration by means of a small trephining opening over the convexity, after the manner of Neisser.

Carrari,[7] in reporting a right-handed temporal lobe abscess which extended backward to the occipital lobe and laterally to the temporal convolution without involvement of the pons, describes what he styles a new type of paralysis alternans, but which I think is largely a result of the complicating meningitis.

Six days after a mastoid operation the patient had rigidity at the nape of the neck, photophobia, positive Babinski and Kernig's signs; the cerebrospinal fluid was turbid, with increased cells and albumin. With a disturbance of consciousness, there were weak conjugate bulbar movements to the right and slow nystagmus. Ten days after the operation, the patient was confused, with euphoria, lying on the left side with flexed knees and hips. Four days later, incision of the dura was made, with evacuation of a spoonful of lemon yellow fluid. Eight days later, the dura was again incised, with the removal of 200 cc. of yellowish bloody

5. Martin, G. F.: Left Chronic Middle Ear Suppuration with Extradural Abscess, J. Laryng. & Otol. **41**:669, 1926.

6. Mayer, C.: A Case of Subdural Abscess at the Convexity of the Cerebrum After Acute Middle Ear Suppuration, Beitr. z. Anat. Physiol., Path. u. Therap. d. Ohres **23**:546. 1926.

7. Carrari, G.: Syndrome of Paralysis Alternans in Otogenic Abscess of the Right Parietal Lobe, Riv. Oto-Neuro-Oftalmol. **3**:469, 1926.

masses. Right-sided ptosis, paralysis of the internal rectus and weakness of the lower facial branch were present. On the left side, the strength of the extremities was diminished, without sensory disturbances; and this weakness gradually increased. Six days later the left patellar reflex was absent; the left tibial reflexes were weak. All sensory qualities of the right frontal and facial half and at the lower extremities were absent. The pupils were wide, without reaction; convergent paralysis and oscillatory nystagmus on looking to the left were present. The papillae were pale and slightly edematous.

(*c*) Cranial Nerve Involvement: Uffenorde [8] thinks that the trifacial pain of Gradenigo's syndrome is due to involvement of the tympanic nerve, which originates in the ganglion petrosum of the glossopharyngeal and constitutes the sensory root of the attic ganglion (trifacial).

Loebell,[9] however, thinks that the relation exists through the perilabyrinthine cells, causing edematous or inflammatory irritation along the edge of the pyramid. In his two cases, meningitic symptoms were absent, as were the manifestations on the part of the abducens nerve.

Sears [10] gives a complete review of the literature and an analysis of the cases of otogenic paralysis of the abducens. He points out that 18 per cent of the patients die. He suggests that anomalous variations in the distribution of branches of the basilar artery in relation to the abducens, influenced by meningitis serosa or cerebral edema, may be a causative factor in the production of the paralysis. Variations in the size of Dorello's canal or increased resistance of the nerve on the side of the affected ear thus exceptionally favor a contralateral sixth nerve paresis.

Ocular paralysis, although generally of little localizating value in suppurative disease of the brain, should always be analyzed, as in Szasz and Richter's [11] case of cerebellar abscess. A paralysis of the left trochlearis, in addition to that of the third nerve, was the cause of the abscess being located in the "anterior median segment" of the posterior fossa, after two unsuccessful explorations of the cerebellar hemisphere itself. The postmortem showed that there were two abscesses, the deeper and more anterior involving the trochlearis at the site where the nerve descends from its nucleus in the roof of the mesencephalon to the base, the nerve being caught, as it were, between the mesencephalon and the anterior and median part of the cerebellar hemisphere.

As far as I know, this is the only case of trochlear paralysis reported in the literature of brain abscess.

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8. Uffenorde, W.: München. med. Wchnschr. **73**:2064 (Dec. 3) 1926.

9. Loebell, H.: Participation of the Cranial Nerves in Suppurations in Perilabyrinthine Foci, Arch. f. Ohren-, Nasen- u. Kehlkopf. **115**:191, 1926.

10. Sears, W. H.: Ann. Otol. Rhinol. & Laryngol. **35**:379 (June) 1926.

11. Szasz, T., and Richter, H.: Otogenous Abscess of the Cerebellum and Paralysis of the Trochlearis, Klin. Wchnschr. **11**:501, 1923.

(*d*) Pulse Differences: Hermann's [12] continuations of Pal's observations of 1920 on pulse differences due to cerebral causes may be of value in that group of temporosphenoidal lobe abscesses which presents uncertain, but not alarming, symptoms subsequent to evacuation. According to his observations on cerebral hemorrhages and softenings (involving chiefly the domain of the internal capsule, the corpus striatum or the posterior part of the temporal lobe), an increased tonus of the radial pulse of the contralateral side occurs in advancing lesions. If this be so, its presence in abscess would be indicative of an extension of the encephalitis.

General Surgical Aids to Cerebral Localization and Prognosis.—(*a*) Ventriculography: Grant,[13] in a discussion on the "Value of Ventriculography," says that of forty patients in whom air was introduced into the ventricles five died, making a mortality of 12 per cent; he adds that in Frasier's clinic they do not feel that a ventriculogram is infallible in diagnosis of the site of the lesion.

(*b*) Ventriculometry: Ney [14] has made 128 ventriculometric readings. He assumes that the normal intraventricular pressure is approximately the same as the pressure of the spinal fluid, namely, about 8 mm. of mercury. He found pressures ranging from 0 to 65 mm. of mercury. Cerebral abscesses give pressures from 25 to 35 mm. of mercury. He says:

> Cerebral abscesses produce about the same degree of pressure elevation as cerebral tumors, and both may be accurately localized by ventriculography. In cerebellar abscesses of otitic origin, the tendency is to produce higher degrees of pressure than in cerebellar tumors because of a frequently associated sinus thrombosis. A lateral sinus thrombosis will usually increase intracranial pressure about 8 mm. Hg.; its presence may be readily determined during ventriculometry by compression of the jugular vein.
>
> In cerebellar abscesses the pressure inciting factors are threefold: (1) abscess; (2) hydrocephalus, produced by posterior fossa pressure; (3) lateral sinus thrombosis in cases of abscess developing from otogenic infections and spreading to the cerebellum through the medium of an infected sinus.

(*c*) Lumbar Puncture: Monroe, in the "Therapeutic Value of Lumbar Puncture in the Treatment of Cranial and Intracranial Injury," [15] directs attention to the baneful influence of increased intracranial pressure on the recovery of the patient. Comparative data obtained on lumbar puncture in the grave and fatal cases of cerebral injury show that the average pressure is nearly two and one-half times higher in the fatal cases.

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12. Hermann, T.: Pulse Differences Due to Cerebral Causes, Wien. klin. Wchnschr. **39**:1209 (Oct. 14) 1926.

13. Grant, F. C.: Ventriculography: Clinical Experience Based on Series of Forty Cases, Arch. Neurol. & Psychiat. **10**:154 (Aug.) 1923.

14. Ney, K. W.: The Measurement of Intracranial Pressure—Ventriculometry, New York State J. Med. **26**:891 (Nov. 1) 1926.

15. Monroe, Donald: Boston M. & S. J. **193**:1187 (Dec. 24) 1925.

Treatment of Encapsulated Brain Abscess.—Dandy [16] cautions against wide drainage, or drainage and irrigation, in brain abscesses, and warns against increasing the effect of traumatism and infection by injudicious therapeutic procedures. He has ventured to treat chronic abscesses by simply evacuating the pus on one or more occasions, utilizing the following procedure, based on personal observations:

> A small perforator opening is made in the bone over the abscess, where it appears to be nearest the dura. A ventricular needle is introduced into the abscess through a tiny nick in the dura. The needle is left in place until pus ceases to drip. The abscess is neither aspirated nor irrigated because of the desire to avoid stirring up the infection by either mechanical or chemical stimuli. The cutaneous wound is tightly closed. The tiny dural opening precludes both a cerebral fungus and a fistulous tract and thereby prevents the introduction of a secondary infection.

This procedure is of proved value only for chronic abscesses. Acute abscesses present a far less hopeful outlook. Here the contraindications to continuous drainage are even greater than for chronic abscesses.

CEREBELLAR ABSCESS

Cerebellar abscesses occur rarely in the first, usually in the second, decade of life, oftener in men than in women and oftener on the right side than on the left. In 80 per cent of the cases, they follow chronic, rarely acute, ear suppurations. Cerebellar abscess is often a secondary process following sinus or labyrinth disease; abscesses occur after acute otitis media, following a thrombophlebitis of the lateral sinus. Purulent labyrinthitis, epidural and cerebellar abscess are a pathologic triad. Whether diffuse or encapsulated, they are always encountered in the vicinity of the ear focus. The inner side of the tense dura is dry, and the bulging cortex is pale. The capsule is formed of vascular and connective tissue, not from glia elements. Between the abscess and the focus the cerebellum appears macroscopically healthy.

Symptoms.—As opposed to temporal lobe abscess, cerebellar abscess is accompanied by more severe occipital pain, persistent vomiting, choked disk, emaciation (trophic disturbance), disturbances of speech and coordination. Cerebellar nystagmus differs from vestibular nystagmus by its duration and excursion to both sides. Nystagmus toward the ear affected by vestibular disease indicates cerebellar abscess; also the persistence of nystagmus after labyrinthectomy. Vertigo appears early, and respiratory disturbances occur only in cerebellar abscess. Caloric nystagmus persists a long time in patients with cerebellar abscess (about eight minutes in some of the cases). A change of direction of the nystagmus (originally turned toward the diseased ear) points to a

16. Dandy, W. E.: Treatment of Chronic Abscess of the Brain by Tapping, J. A. M. A. **87**:1477 (Oct. 30) 1926.

complication of the labyrinthitis by a cerebellar abscess. Testing of the patient's weight, psychic changes and demonstration of homolateral paralyses are of importance in the diagnosis of cerebellar abscess. A single conclusive symptom does not exist. Patients with cerebellar disease totter with the eyes open and have vertigo without nystagmus.[17]

Latency of Symptoms of Cerebellar Abscess.—Ferreri [18] contributes a compilation of the symptoms possibly present in cerebellar abscess. Distinction is made between (1) symptoms referable to compression (analogous to those furnished by temporal lobe abscess or tumor formation) and (2) focal symptoms. There are no focal symptoms for the cerebellum, as for the cerebrum, but only phenomena of a functional alteration. The cerebellar centers regulate motion by maintaining the limbs in a state of constant tonus; when a center is damaged, the muscular tonus dependent on the healthy center acquires ascendency. All rapidly developing abscesses lead to compression phenomena with predominance of bulbar symptoms which suggest a basilar abscess and often prove misleading. There are (3) changes of the specific function of the cerebellum, cerebellar ataxia and adiadochokinesis. Notwithstanding the large number of pathologic signs, the diagnosis remains so difficult that as yet it is not made in 90 per cent of the cases. The patients are often first seen when the local phenomena are concealed by the general symptoms and the increased intracranial pressure. The diagnostic difficulties are illustrated by four highly instructive clinical histories.

On the other hand, the cerebellar symptoms may be so outstanding as to admit of no other interpretation, as in Lemaitre and Aubin's [19] case of a woman who had suffered for twenty-five years from otorrhea and who presented paralysis of the acousticofacial nerve, anesthesia of the trigeminus and cerebellar symptoms. A cholesteatoma the size of an egg was found, which had perforated the posterior surface of the pyramid and destroyed the nerve trunk.

Technic of Operating for Cerebellar Abscess.—Towne [20] describes a method of approach from the median line which I have found useful in suspected cerebellar abscess. Towne's incision when combined with a gradually enlarged horizontal incision toward the affected petrous bone and associated with the primary removal of the bone from the median line and subsequent approach toward the mastoid, not only allows the

17. Bocache, L.: Otitic Cerebellar Abscesses, Ann. d. méd. Nav. e Col. **2**:76, 1926.

18. Ferreri, G.: Latent cerebellar abscesses, Arch. Internat. d. laryngol. otol. rhinol. **5**:897, 1926; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **9**:723, 1926.

19. Lemaitre and Aubin: Cholesteatoma of the Cerebellar Fossa, Oto-rhino-laryngol. Internat. **10**:334, 1926.

20. Towne, E. B.: Exploration of the Cerebellum, Surg. Gynec. Obst. **42**:204 (Aug.) 1926.

operation to be conducted through a clean field, but also, if the cerebellum be retracted materially, diminishes the distance to the posterior surface of the petrous pyramid.

Lemaitre [19] describes his well known method of walling off the subarachnoid space by gradually enlarging drainage tubes, which he apparently thinks are as feasible in cerebellar as in temporosphenoidal abscess regardless of the manifestly different mechanical, anatomic and physiologic factors.

MENINGITIS

Classification.—In an attempt at classification of meningitis of otitic origin, Aboulker [21] saw that meningeal inflammations are distinguished: (1) according to their course, into acute and chronic; (2) according to their clinical manifestations, into diffuse meningitis and those with local symptoms. The acute diffuse forms may be septic or aseptic, and are, for the most part, the sequelae of an acute or chronic suppurative otitis. They usually assume a violent course, and signs of their otitic origin are absent. Acute meningitis with intracranial pressure symptoms may present: (1) cerebral (general and localized); (2) cerebellar and, (3) cerebro-erebellar, which frequently simulate the picture of brain abscess. The chronic meningitides, on the other hand, which are usually the sequelae of a chronic purulent (encapsulated) otitis, resemble intracranial tumors when associated with pressure symptoms, and when diffuse, present the picture of chronic meningoencephalitis. In a given case, the character of the meningitis depends on the site of the primary infection. The author distinguishes between a "temporalitis antro-tympanica," "antro-tympanomastoidea," "labyrinthica, squamosa, condyloidea," etc., and a corresponding meningitis temporosphenoidalis, temporoparietalis, retropetrosa (with labyrinth symptoms), ponto-cerebellosa, etc., according to whether the distribution of the infection from the tympanic cavity takes place by way of the anterosuperior or postero-inferior paths. In no form of temporalitis and temporo-encephalitis can it be determined in advance at what point the entrance into the cranial cavity will occur. Irritative symptoms of the cranial nerves often announce the advance of the infection in cases in which symptoms of a persisting disease of the temporal bone are no longer demonstrable.

Otitic Meningitis in General.—Hinsberg [22] reports "on 137 cases of otogenic meningitis observed from 1903-1925 in the Breslau Oto-RhinoLaryngological Clinic." Meningitis of labyrinthine origin is divided by him into (1) meningitis present prior to any operation on

21. Aboulker, H.: Rev. d' oto-neuro-ocul. **4**:405, 1926.

22. Hinsberg, V.: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres. **23**:124, 1926.

the middle ear. He is of the opinion that with acute labyrinthitis appearing in the suppurative stage of acute otitis media, the labyrinth should be opened as soon as possible. (2) Postoperative meningitis has several subdivisions: (*a*) meningitis after extraction of polyps in labyrinthitis; (*b*) postoperative labyrinthitis and meningitis after radical operation, in which the infected labyrinth should be opened; (*c*) postoperative meningitis as a sequel of labyrinthitis which was circumscribed before the operation and has become diffuse as a result of the operation; (*d*) postoperative meningitis after opening of the labyrinth; (*e*) postoperative meningitis due to radical operation without opening of labyrinth in diffuse labyrinthitis. Meningitis derived from the labyrinth begins, as a rule, in the posterior cranial fossa and in the majority of the cases remains limited to it for some time. There is also (*f*) meningitis after radical operative injury to the labyrinth. Meningitis in some cases is derived from extradural abscess or from a bone focus which has been overlooked at the time of the middle ear operation; or it may follow injury or incision of the dura. Meningitis may occur in brain abscess, be associated with sinus thrombosis, follow fracture at the base of the skull, or be of obscure origin. All these various subdivisions are illustrated by brief clinical observations. The author reports thirty-two recoveries among his 137 cases (23.3 per cent). His material confirms the known fact that labyrinthogenic meningitis has a somewhat more favorable prognosis than meningitis of different origin. The prospects are better in proportion to the timely recognition and removal of the primary focus.

Mucosus Meningitis.—Urbantschitsch [23] describes in detail eight cases of mucosus otitis, with autopsy observations. On the basis of personal and other observations, he emphasizes the latency of the process, the difficulty of diagnosis and the frequency of early errors of diagnosis. After the meningitis distinctly manifests itself clinically, the course is usually very rapidly fatal. Recovery is exceedingly uncommon. Among twenty-two observations, the author notes only one cure, in a child, aged 2. The low temperatures during a long period of time are noteworthy, since the patients go about without suspicion of a grave disease. Men and persons of advanced age seem to be predominantly affected. But young children may be attacked, and the prognosis in such instances seems actually to be more favorable. Among his eight patients, six had sinus thrombophlebitis. This is not mentioned elsewhere. The mastoid process must be opened as promptly as possible. The operation is performed bilaterally by the author in children under 2 years of age; also when there is apparently no inflammation in one ear, because the ear

23. Urbantschitsch, E.: Mucosus meningitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **16**:129, 1926.

disease is usually bilateral and often cannot be positively recognized. The intraspinal administration of streptococcus serum is recommended. The greatest importance is attached to early diagnosis by timely spinal puncture and bacteriologic demonstration of the pathogenic contents, as the diagnosis from examination of the tympanic membrane in children is misleading.

Paths of Infection.—The discovery of the path of infection in fulminating meningitis, which is so frequently associated with or preceded by an acute pneumococcic or streptococcic otitis, is of first importance, as on its demonstration the future of all surgical or chemical treatment must depend. From the experimental studies of Weed and his associates in 1914-1920, it would appear as if in the presence of a blood stream infection the pia-arachnoid spaces of the cortex were first invaded by the micro-organisms, and I obtained postmortem evidence that in fulminating pneumococcic meningitis there is a general distribution of micro-organisms through all the accessory sinuses of the nose.[24]

Klepacki 's [25] carefully conducted histologic examinations in two cases of meningococcic meningitis may consequently be of otologic value, as he demonstrated that in fulminating meningococcic meningitis there are at first isolated patches of inflammation in the blood vessels of the choroid plexus from circumscribed purulent foci of blood stream origin. From the choroid plexus the infection extends to the ventricles and later to the meninges of the cortex.

Healing in Meningitic Labyrinthitis.—Kasai [26] performed experiments at the Ear, Nose and Throat Clinic of the University of Okayama, Japan. Two loopfuls of an agar culture of moderately virulent streptococci were distributed in 5 cc. of physiologic salt solution, and fifteen rabbits were injected with this bacterial emulsion (one-quarter of a loopful) into the subarachnoid space, between the occiput and the atlas. Nine animals died of meningitis, and six recovered, but became deaf and showed functional disturbances of the vestibular apparatus. These six rabbits were kept under continued clinical observation for from two and a half to six months. At the end of this time their auditory organs were examined histologically. The observations were as follows: The healing processes of the labyrinth consist, in the first place, in organization of the inflammatory products in the labyrinth spaces, by connective tissue, followed by ossification, so that

24. Eagleton, W. P.: Operative Treatment of Suppurative Meningitis; Case 1: Acute Fulminating Pneumococci Type III Meningitis, Tr. Am. Otol. Soc., 1921.

25. Klepacki, Witold: Metastatic Foci in the Choroid Plexuses as Starting Point of Ventriculitis and Meningococcus Meningitis, Presse méd. **34**:1096, 1926.

26. Kasai, T.: Experimental Studies on Meningogenic Labyrinthitis. Curative Processes in Meningogenic Labyrinthitis, Japan M. World **6**:144, 1926.

finally the cavities are, for the most part, filled by new-formed bone. The tendency to new bony formation is most marked in the cochlea, next in the semicircular canals and the vestibule. The distribution of the inflammation from the subarachnoid space into the labyrinth takes place chiefly through the cochlear aqueduct. The vestibular aqueduct plays no part in this connection. The labyrinthitis may rupture from the apical portion of the cochlea into the middle ear. The utricular otoliths are without relation to caloric and rotatory nystagmus. On the other hand, the utriculus and sacculus participate in the labyrinthine cervical reflexes, while the semicircular canals are of no significance for these reflexes.

Localized Meningitis.—Jenkins [27] reports two cases of what he terms "cystic serous meningitis" associated with recurring otitis (in spite of mastoid operations). The symptoms were gradual general debility, hysteria, giddiness, vertigo, tinnitus and headache. In one the ear was dry at the time, and in both the labyrinth was functioning. Incision of the dura behind the sinus and posterior to the labyrinth evacuated a large quantity of cerebrospinal fluid; the cerebellum did not come into view, and the spatula entered a large space. The symptoms immediately disappeared in both cases. It is unfortunate that no examination was made of the evacuated fluid. The cases apparently belong to the type described by Horrax as pia-arachnoiditis.

Cerebrospinal Fluid in the Diagnosis of Meningitis.—Greenfield,[28] in an important communication on the *chemistry* of the *cerebrospinal fluid in otitic meningitis,* says that normally the fluid in the ventricles is saline, contains no cells and has only one part in 20,000 of protein. It is possible that even this trace is not present at the time of secretion. Sodium chloride is constantly present and is in much larger amount in the fluid than in the blood, being steadily between 700 and 750 mg. per hundred cubic centimeters. Glucose is present in from 50 to 60 per cent of the blood sugar content and so remains in all conditions with the exception of meningitis. Urea is in practically identical concentration in the blood and in the cerebrospinal fluid. Any rise in the blood urea above 50 mg. is associated with a corresponding urea concentration in the fluid. No matter how much the chloride content of the blood is increased, it has no influence on its percentage in the cerebrospinal fluid. Therefore, normally, cells of the choroid plexus act as a selective barrier between the blood and the fluid. When there is widespread meningeal inflammation, however, the chemical composition of the cerebrospinal fluid approximates that of the blood. Chlorides are reduced, and the other acid radicals increased. Glucose tends to disappear, probably owing to the putrefactive action of the bacteria or to the elements set

27. Jenkins, G. J.: J. Laryng. & Otol. **41**:781 (Dec.) 1926.
28. Greenfield, J. G.: J. Laryng. & Otol. **41**:785 (Dec.) 1926.

free in the disintegration of the leukocytes. Greenfield believes that it is injury to the choroid plexus by the inflammation of the meningitis that causes some of the changes in the fluid by preventing its selective action on the constituents of the blood. For so long as the meningitis is limited to a small area, viz., around the petrous bone, the percentages of inorganic salts in the fluid remain unaltered, whereas, when the meningitis becomes more widespread, the percentages tend to approximate those of the blood. In a previous paper he stated that the approximation of the chloride content of the fluid was an unfavorable prognostic omen, while continuance of a higher chloride percentage than in the blood signified that the barrier is still intact, and consequently reparable. This frequently occurs in spite of a turbid purulent fluid. The fibrin coagulum indicates that the fibrinogen of the blood is allowed to pass the barrier. He concludes that prognosis in meningitis can no longer be based on the bacteriologic examination of the fluid alone. The chloride percentage must be estimated.

On the other hand, Bertoin,[29] in discussing "Aseptic Purulent Meningitis of Otitic Origin," says that etiologically the purulent fluid represents not so much the passage of the white blood corpuscles in the arachnoid around the pus focus as the toxic effects on the cerebrospinal fluid. Or it may originate in agglutinations of the meninges in the neighborhood of the primary focus or from a circumscribed purulent meningitis. Examination of the fluid obtained on spinal puncture alone is insufficient, but when, for example, the reduction capacity of sugar, which disappears on leukocytosis, returns to normal, it has favorable significance. An entire series of fatal outcomes of aseptic meningitis have been observed. The treatment consists in local intervention assisted by repeated spinal punctures and injection of serum or antitoxic bodies. Incision of the meninges should be reserved for exceptional cases.

Lactic Acid in Fluid.—Lactic acid in the cerebrospinal fluid has a normal proportion of about 11 to 27 per hundred cubic centimeters, the percentage of lactic acid in the blood varying from 60 to 96 per cent. In one case of meningitis and in another of brain abscess, Glaser[30] says there was an absolute increase in the lactic acid of the spinal fluid. The result of his series leads to the conclusion that when the percentage ratio of spinal fluid lactic acid to blood lactic acid is in excess of 100, a pathologic condition of the central nervous system can be suspected.

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29. Bertoin, R.: Aseptic "Puriform" Meningitis of Otitic Origin, J. de méd. de Lyon **7**:433, 1926.

30. Glaser, Jerome: The Lactic Acid Contents of Cerebrospinal Fluid, J. Biol. Chem. **20**:539 (Aug.) 1926.

Hemorrhage into the Pia-Arachnoid, Simulating Meningitis.—Skillern,[31] under the caption of "An Unusual Ethmoidal Finding," reports a case in which a probe was passed by way of a dehiscence in the posterior ethmoid capsule or an unusually large posterior ethmoid foramen into the brain substance. The patient recovered. One month later he was suddenly seized with an intense headache, slow pulse, exaggerated reflexes and general restlessness of meningitis. Lumbar puncture gave bloody fluid not under pressure. He died in coma within thirty-six hours.

Autopsy gave no evidence of old or recent meningitis, but the brain was bathed in considerable blood. Its surface was hemorrhagic, and in the right cerebral hemisphere there was a softened area. A cut section of the area revealed a group of telangiectatic blood vessels with relatively thin walls and filled with clotted blood. The microscopic picture suggested a circumscribed hemangioma of the cavernous type. Hemorrhage filled the ventricles and the subarachnoid and subdural spaces. No dehiscence or tract through which the probe had passed could be found.

It is my opinion that Skillern's case belongs to that group of spontaneous pia-arachnoid hemorrhages into the basal cysterna first described by Symond [32] in 1923, by Riddoch and Goulden [33] in 1925 and by Calhoun [34] during the past year. Since becoming familiar with the condition, I have seen three of these cases, all diagnosed as meningitis because of the elevated temperature, stiff neck and headache. Lumbar puncture revealed a bloody, but sterile fluid. The stiff neck, headache and temperature are believed to be due to the reaction from the bloody effusion in the basal cysterna. Postmortem examination in one of my cases demonstrated an area of bloody fluid in the base. Massive lumbar puncture may be fatal, increasing the hemorrhage by suddenly lowering the pressure.

Operative Treatment of Meningitis.—During the past year I have contributed two papers in an effort to establish a philosophy of meningitis by means of which it is hoped that the surgical treatment may be conducted according to some definite policy [35] depending on (*a*) the route of infection, (*b*) the stage at which seen and (*c*) the type of infection.

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31. Skillern, S. R.: An Unusual Ethmoidal Finding, Ann. Otol. Rhinol. Laryngol. **35**:71, 1926.

32. Symond, C. P.: Contributions to the Clinical Study of Intra-Cranial Aneurisms, Guy's Hosp. Rep. **73**:139 (April) 1923.

33. Riddoch, G., and Goulden, C.: On the Relationship Between Subarachnoid and Intraocular Hemorrhage, Brit. J. Ophth. **9**:209 (May) 1925.

34. Calhoun, F. P.: Ocular Symptoms in Subarachnoid Hemorrhages, Tr. Sect. Ophth. A. M. A., 1926.

35. Eagleton, W. P.: Philosophy of Meningitis, Laryngoscope, October, 1926; Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926.

Richards,[36] in an important surgical discussion of the "petrous pyramid," reports a cured case of streptococcic meningitis by drainage of the posterior fossa through the internal auditory meatus, associated with repeated lumbar punctures. He also describes the small, rapid irregular pulse, embarrassed respiration—which may assume a Cheyne-Stokes' character—anxious expression, cold extremities, pinched features and marked restlessness which follows too sudden escape of cerebrospinal fluid from fracture of the modiolus. Watson-Williams [37] urges early operation, even before a positive diagnosis of septic meningitis is made. His summary is as follows: In every case of meningitis in the presence of otorrhea, the question of aural operation should be considered at an early stage. The decision as to operation may have to be made before it is possible to determine whether the meningitis is septic: to wait until a doubtful case is certain is to throw away the best chance of success. Therefore, the value of lumbar puncture in settling the question is limited. If the cerebrospinal fluid shows manifest changes, that may be an additional reason for operating; but, obviously, the physician hopes, by basing the decision on physical signs, to anticipate this stage. Lumbar puncture is of great value to the surgeon in determining the type and scope of the operation. For the same reason, tests of the labyrinth should never be omitted. Delay of a few hours may well turn the scale from success to failure.

Manasse [38] states that when the primary focus is situated in the vicinity of the cerebral meninges, it should be removed. When the dura is diseased it should be widely incised, the wound margins drawn apart, the brain mass pushed back with a small spatula, and a broad flat gauze strip pushed under the brain. The dressings are changed every two days. Spinal puncture is performed every day or on alternate days. When the primary focus is not situated in the vicinity of the dura, spinal or suboccipital puncture is relied on. When the meningitis presents local symptoms pointing to the position of the effusion, permanent drainage should be applied to this point.

Aloin [39] discusses the causes, symptoms and prognosis of "lymphangitic" meningitis. He considers it as usually of tuberculous origin, but also other chronic processes—cholesteatoma, syphilis, herpes, uremia and (in infants) typhoid fever—may enter into consideration. As

36. Richards, J. D.: Petrous Pyramid; Its Surgical Anatomy, Am. J. Surg. **2**:11 (Jan.) 1927.

37. Watson-Williams, E: The Operative Treatment of Meningitis (four illustrative cases), Bristol Med.-Chir. J. **43**:91, 1926.

38. Manasse, P.: Operative Treatment of Induced Meningitis, Forschr. d. Therap. **2**:309, 1926.

39. Aloin, H.: Lymphocytic Meningitis of Otitic Origin, Contribution to the Surgical Treatment of Meningitis, J. de méd. de Lyon **7**:579, 1926.

initial symptoms, he emphasizes trigeminal neuralgia, intestinal and vesical disturbances (contractions) and spontaneous pain of the external occipital protuberance, which often radiates along the entire vertebral column. The lymphocytic form of meningitis is to be interpreted as the proof of a slow chronic process and therefore has a favorable prognosis. Operative therapy would be useless when the infectious focus is inaccessible to good drainage (apex of petrous bone), or when the meningitis is already diffuse. Under such conditions, opening of the cisterns or irrigation with chemical agents, etc., would be an experiment not particularly promising success. The author recommends for the support of the organism polyvalent streptococcus or staphylococcus serum, and in addition from 250 to 500 cc. of simple or glycosed salt solution daily. His views on the advantage of cistern drainage are pessimistic. Two cases of recovery are reported.

Caliceti [40] reports a case of otitic meningitis in which the patient was cured by repeated lumbar punctures and intravenous injections of methenamine. The mastoid was drained. No dural incision was made. There is no mention of bacteria in the fluid. Kolmer [41] demonstrated that lavage from the ventricles through the occipito-atloid region, if performed early enough, cured experimentally induced meningitis, which had been produced by the intracisternal injections of virulent pneumococci and streptococci. Serums and other chemicals had little effect.

McAskill [42] enumerates the methods of treatment described in the literature, notably the investigations of Ayer on the mutual relation between the pressure in the cerebrospinal fluid and the cerebral veins, and the effect of injection of chemical agents into the cisterna magna. Three patients with meningitis were treated by him with mercurochrome-220 soluble, with favorable results. One case is reported in detail, in which the patient reacted poorly to an intravenous injection of mercurochrome, but promptly showed improvement after an injection into the cisterna magna, and was cured by three more cistern injections and one intraspinal injection. The lumbar fluid contained a "gram-positive organism."

Metzger [43] used intravenous injections of sodium iodide (from 10 to 20 cc. of a 10 per cent solution) in the treatment of *septic meningitis* (of anginal origin). He describes the dangers of cisterna puncture from injury to a pial vessel, which he states is especially apt

40. Caliceti, P.: Some Comments on a Case of Otogenic Purulent Aseptic Meningitis, Ann. di Otol. Rinol. Laring. **2**:209, 1926.

41. Kolmer, J. A.: Chemotherapy and Serum Treatment of Pneumococcus and Streptococcus Meningitis, Arch. Otolaryng. **3**:481 (June) 1926.

42. McAskill, J. E.: Treatment of Purulent Leptomeningitis Secondary to Otitis Media, Ann. Otol. Rhin. Laryngol. **35**:502, 1926.

43. Metzger, E.: Zur Therapie der septischen Meningitis (treatment of septic meningitis), Deutsche med. Wchnschr. **52**:1902, 1926.

to happen in meningitis because the vessels are full and easily injured. Immediately after the puncture the patient had a right-sided ptosis (lasting a half hour), nystagmus in all directions and diplopia. Recurring attacks of scintillating scotoma lasted for several days. The nystagmus (which was to the right, left and upward) gradually subsided, but could still be demonstrated some time after the accident.

Encouraged by Metzger's favorable results, Schmidt [44] treated a patient with septic meningitis by intravenous injections of "septiod," as already recommended by Fleischmann in 1922 for the treatment of purulent meningitis by way of the blood. The doses of septiod were increased from 20 to 40 cc. daily, until 350 cc. was given in all. At the same time, spinal puncture was made every other day, from 20 to 30 cc. of fluid being withdrawn according to the pressure, which fluctuated between 350 and 420. Altogether, four punctures were made in the course of six days. The condition improved after the fourth spinal puncture; the patient made a good recovery. No bacteriologic report was given. It may have been a meningococcus infection.

TUMORS (ACOUSTIC AND PINEAL) CAUSING CENTRAL DEAFNESS

Barrett [45] reports the clinical history and autopsy of a "case of spinal rhinorrhea" accompanying multiple tumors of the left acoustic nerve (Cushing). Although not so stated in the report, the rhinorrhea was apparently the result of distention from continued pressure of the perivascular spaces surrounding the olfactory nerves, probably precipitated by the removal of support from a previous submucous resection of the septum.

Bilateral Central Deafness.—Horrax [46] reports a series of "tumors of the pineal body," five of which were associated with a greater or less degree of central deafness. This deafness was undoubtedly due to pressure on the central connections in the inferior colliculi. Bilateral deafness associated with inability to move the eyes upward (from involvement of the anterior colliculi) should indicate a diagnosis of tumor of the pineal region. In reading the clinical history I am impressed with the usual lack of complete and systematically conducted examinations of the vestibular and cochlear functions, even in tumors manifestly involving these senses.[47] It speaks for a closer cooperation between the surgeon and the otologists.

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44. Schmidt, K.: Therapy of Septic Meningitis, Deutsche med. Wchnschr. **52**:2079, 1926.

45. Barrett, J.: M. J. Australia **2**:182 (Aug. 7) 1926.

46. Horrax, G., and Bailey, P.: Tumors of the Pineal Body, Arch. Neurol. & Psychiat. **13**:423 (April) 1925.

47. Throckmorton, T. B.: Glioma of Pons Varolii with Autopsy Report, J.A.M.A. **87**:1731 (Nov. 20) 1926.

Towne [48] reports three cases of acoustic nerve tumor diagnosed by a roentgenogram, a new position being used. He claims that tumors of the eighth nerve arise in the internal auditory canal and erode the petrous bone. There is a long interval, averaging about five years, between the onset of the initial auditory symptoms and the appearance of the signs which make the diagnosis certain. During this interval the diagnosis cannot be made, because tinnitus and nerve deafness may be due to other causes. Previous attempts to show the erosion of the petrous bones in roentgenograms with lateral projection have been unsatisfactory. In all three cases of verified acoustic nerve tumor examined roentgenographically with the occipital projection (Chamberlain's technic), extensive erosions of the petrous bone on the side of the tumor were clearly shown. It seems probable that the use of this technic during the period when only auditory symptoms are present may lead to earlier diagnosis and surgical treatment.

VESTIBULAR REACTIONS IN NONSUPPURATIVE LESIONS

Eiselsberg [49] regards "rotation vertigo" as of localizing value in the diagnosis of tumors of the posterior fossa. Brunner,[50] from his association with Eiselsberg's clinic, contributes an extensive paper on the "Results of Clinical Functional Tests of the Ear in Diseases of the Medulla Oblongata and the Cerebellum" in nonsuppurative lesions.

Of *multiple sclerosis,* he says that auditory symptoms are absent, but vestibular symptoms are common. Labyrinthine vertigo occurs as (1) rotary, or (2) tactile vertigo. Rotary vertigo is frequent in incipient cases. Brunner calls especial attention to the association of rotary nystagmus and visual disturbances in the early stages of multiple sclerosis. The patients frequently have a sensation of being drawn to one side, and they may fall to that side. Lateral propulsion may occur independently of rotary vertigo or spontaneous nystagmus. Spontaneous nystagmus is a frequent symptom of multiple sclerosis, but when not present the hyperirritability can generally be demonstrated by sudden movements of the head, producing a varying degree of nystagmus. Spontaneous past pointing was not noted by Brunner, but Romberg's sign is usually present. Caloric tests show hyperirritability.

Nonsuppurative encephalitis, that is, (1) encephalitis lethargica or epidemic encephalitis, and (2) polioencephalitis inferior, which includes pontile and cerebellar encephalitis (Redlich) and acute hemorrhagic polioencephalitis superior (Wernicke), may give symptoms of cochlear

48. Towne, E. B.: Erosion of the Petrous Bone by Acoustic Nerve Tumor, Arch. Otolaryng. **4**:515 (Dec.) 1926.

49. Eiselsberg, A.: Problems of Cerebral and Spinal Surgery, Arch. f. klin. Chir. **142**:205, 1926.

50. Brunner, H.: Ztschr. f. d. ges. Neurol. u. Psychiat. **37**:145, 1926.

or vestibular involvement in varying degrees, depending on the position of the lesion. In a general way, labyrinthine rotary vertigo is rare in epidemic encephalitis, but spontaneous nystagmus is common. The Romberg sign is apt to be present, and ataxic disturbances in walking are especially common. In Redlich's type, symptoms of eighth nerve involvement play an important part. There are only a few recorded otologic observations in the Wernicke type, as apparently the patients have not been submitted to a special aural examination.

Diseases of the posterior inferior cerebellar artery give outstanding signs of vestibular involvement because of the sudden onset of the vascular disturbance. The accompanying severe vertigo is either typically rotary in type or manifests itself as a tottering sensation. Although the vertigo gradually diminishes, it rarely disappears entirely and is apt to recur in milder attacks. The degree of spontaneous nystagmus in posterior inferior cerebellar arterial disease depends on whether the case is observed early in the attack or not. It differs from the spontaneous nystagmus of labyrinth disease in continuing toward the affected side, while labyrinthine nystagmus, after the subsidence of the attack, is apt to be toward the healthy side. Brunner thinks this is because in the arterial disease, the nystagmus results from involvement of the spinal root of the acoustic nerve or of fibrillar systems directly connected with this brain area (arcuate fibers, vestibulospinal tract). The spontaneous nystagmus is usually horizontal rotary, rarely pure rotary.

Vertical nystagmus has not been noted. Equilibrium disturbances are equally common with nystagmus; the patient falls toward the affected side, viz., in the direction of the quick component of the nystagmus, thus differing from the falling in labyrinth disease, in which the falling is in the direction of the slow component. Disturbances of equilibrium gradually disappear, but may be demonstrated later by quick turning of the head, etc. Labyrinth irritability to all forms of stimulation is preserved. The acoustic function shows no disturbance.

TUMORS

1. *Angle Tumors.*—Angle tumors included several varieties having only their position (in the pontile cerebellar angle) in common. Brunner divides them into (*a*) metastatic sarcomas and carcinomas; (*b*) tumors penetrating the angle from the throat or parotid gland by way of the jugular fossa; (*c*) tumors springing from the meninges (endotheliomas) or from the blood vessels (peritheliomas); (*d*) tumors that pass from the cerebellum or the pons into the angle (gliomas); (*e*) tumors of the nerves themselves (neuromas).

Brunner says that while a review of the aural symptoms shows that none taken by itself is absolutely conclusive for a diagnosis of angle

tumor, still a combination of homolateral deafness, nonirritability for all stimuli and distention of the auditory meatus will permit of the diagnosis. But even here a differential diagnosis of the type of tumor is difficult, but repeated examinations over a prolonged period will generally determine whether the tumor originates in or gradually invades the angle.

(Brunner apparently does not recognize the importance in this connection of the sign of increased intracranial pressure afforded by an "absence of irritability to the cold caloric in the upright position of the contralateral side," which was first described by me in 1912.)[51]

Cerebellar tumors, as a rule, do not present unilateral deafness or unilateral complete nonirritability of the homolateral side, although Brunner says there are exceptions to this rule.

Tumors of the angle may be distinguished from a pons tumor, as the latter not only lacks auditory manifestations, but is generally associated with anomalies in the position of the eyes, sensory disturbances and sometimes choked disk, but the differential diagnosis may meet with great difficulty when the pons tumor has invaded the angle. The differential diagnosis between tumors of the acoustic nerve and of the cranial base or the gasserian ganglion, which invade the angle, can only exceptionally be made by the ear examination alone, but is occasionally possible by other neurologic signs and a study of a chronological history.

The differential diagnosis between multiple sclerosis and a tumor of the angle may be difficult; the outstanding aural symptoms generally subside in the former, but increase or remain stationary in the latter, while choked disk rarely occurs in multiple sclerosis.

Brunner says:

> American authors (Eagleton, Fisher, and others) have pointed out that in brain pressure, such as is found primarily in tumors of the posterior cranial fossa, the irritability relations are changed in so far as the horizontal canals respond, while the reactions from the vertical canals are absent, as a whole or in part.

Brunner was unable to test these statements, as it is still problematic whether the stimulation of isolated semicircular canals is at all practicable; and an examination on the revolving chair, by means of which this test would be most feasible, is not devoid of danger in brain tumors and must therefore be restricted as much as possible. Caloric tests, as utilized by some American physicians for this examination, seem to him entirely inappropriate for obtaining reliable conclusions as to the behavior of individual semicircular canals.

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51. Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin; Report of 3 Cases with a Previously Undescribed Aural Condition, Tr. Am. Otol. Soc., 1912.

2. *Cerebellar Tumors.*—Brunner's report is a compilation of a careful examination in forty cases of cerebellar tumors, either personally observed or taken from the literature. The cochlear function is not affected unless the tumor invades the angle. This may occur in cerebellar tuberculosis. In cerebellar tumors vertigo and its associated conditions are especially apt to be present. Labyrinth hyperirritability, as well as nystagmus to the side of the dead labyrinth, frequently seen in cerebellar abscess, is explained by Brunner as the effect of pressure. Such hyperirritability is also found in cerebral tumors and in internal hydrocephalus.

As regards falling in cerebellar tumors, Brunner says that no reliable conclusions as to the localization of a tumor can be drawn from the direction of spontaneous falling, but it may be stated with some degree of certainty that genuine cerebellar falling takes place predominantly in the sagittal plane, while falling toward the side is referable to a disease of the labyrinthine system. The patients fall toward the diseased side in a long series of cerebellar tumors, but this is not true in all of the cases. The labyrinthine falling reactions in cerebellar tumors are still more complicated than spontaneous falling. Among ten tumors which were predominantly restricted to the hemisphere and in which the falling reactions were carefully investigated, six showed typical falling reactions; in four cases, these reactions were atypical. Concerning disturbances of gait in cerebellar tumors, these possess, from the practical viewpoint, essentially the same significance as disturbances of equilibrium, but their central mechanism is still more complicated than that of disturbances of equilibrium.

Brunner utilizes the pointing tests of Bárány for the confirmation of a diagnosis only when supported by other clinical symptoms, but does not consider them when they cannot be fitted into a clear clinical picture.

The condition of the ear in frontal lobe tumors may vary greatly, and the difficulty of the differential diagnosis from cerebellar tumors may prove considerable.

3. *Tumors of the Pons and the Medulla Oblongata.*—In the presence of tumors of the pons and the medulla oblongata, normal auditory conditions are noted even when portions of the central cochlear system are positively involved. On the other hand, unilateral deafness may result when the cochlearis trunk is invaded by the tumor.

In the examination of the ocular movements a distinction must be made between spontaneous abnormalities in the position of the eyes (deviations, et cetera) and spontaneous nystagmus. The former are characteristic of pontile tumors; the latter occurs especially in diseases of the medulla. Only acute conditions of the vestibular nuclei, vascular in character, give rise to conjugate deviation of the eyes, which is transi-

tory. Spontaneous nystagmus is not characteristic in tumors of the pons or medulla. Sometimes it is present toward both sides and again absent.

Disturbances of equilibrium and of the pointing test are absent in the majority of tumors restricted to the pons. Induced labyrinthine reactions—marked retardation of the nystagmus, absence of the rapid component—appear in partial lesions of the posterior longitudinal bundle. In a general way it may be stated that in partial lesions of the posterior longitudinal bundle, the voluntary ocular movements are affected much earlier than those of labyrinthine origin.

Investigations on Galvanic Nystagmus in Brain Disease and Disturbances of Consciousness.—Rosenfeld,[52] demonstrates that there are great individual fluctuations in the strength of the current required to produce rotary nystagmus in normal subjects. Consequently, caloric stimulation and that obtained from rotation are, in a general way, more efficient than galvanic stimulation, especially as in some cases reactibility of the vestibular apparatus was still present when it was impossible to elicit nystagmus and associated phenomena by the galvanic current. However, in reading the report I was impressed with the fact that galvanic stimulation may be of great diagnostic value, especially in hysterical and semicomatose or comatose states. Rosenfeld demonstrates that in disturbances of consciousness the susceptibility of the central nervous system to the galvanic current is considerably diminished. He claims that there is a parallelism between the susceptibility of the central nervous system to the galvanic current and to disturbances of consciousness. Consequently, it would appear to me that in semicoma from abscess or in the restlessness of meningitis, galvanic stimulation giving a slow deviation of the eyes to the side of the anode might be of value in demonstrating that the central vestibular apparatus was still functioning in those cases in which the labyrinth is dead, or in which caloric stimulation is impossible or not advisable.

Fox and Holmes [53] write on "Optic Nystagmus and Its Value in the Localization of Cerebral Lesions." Forty patients with cerebral tumor were examined, the site of the lesion being verified by operation or postmortem in twenty-four, besides numerous cases of cerebral injury, vascular disease and cerebral syphilis. Seven cases are reported in detail. The results are of great localizing value for tumors, and if confirmed, should be of distinct aid in the localization of abscess. The authors make the definite statement that a unilateral lesion of the forebrain can disturb lateral ocular nystagmus. If the lesion lies in the

52. Rosenfeld, M.: Klin. Wchnschr. **5**:1815, 1926.
53. Fox, J. C., and Holmes, Gordon: Brain **49**:333, 1926.

right hemisphere, clockwise rotation of the examining drum—that is, when rotated from right to left—evokes a nystagmus consisting of slow deviations of the eyes to the left and quick jerks to the right—in other words, a normal nystagmus to the right. But *anticlockwise rotation has no effect or produces a less regular or less pronounced nystagmus to the left.* They state that it seems that nystagmus to the opposite side is affected when a lesion lies in the supramarginal or angular gyrus, in the adjacent portion of the parietal and temporal lobes, in the posterior end of the second frontal convolution or along a line joining this with the angular gyrus. They believe that their observations tend to support Stenver's hypothesis that reflex centers for optic nystagmus lie in the occipital lobe and in the second frontal convolution, and that these are connected by a reflex path which runs through the white matter of the hemisphere.

News and Comment

EXAMINATIONS GIVEN BY THE AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology conducted an examination at Washington, D. C., May 16 and 17, and at Spokane, Wash., June 4. Of the 142 men examined at Washington, D. C., 119 were passed and 2 failed to pass; in Spokane, 46 passed and 6 failed.

The next examination will be held in Detroit on Sept. 12, 1927. Applications for examination should be sent to Dr. H. W. Loeb, Secretary, 1402 South Grand Boulevard, St. Louis.

Abstracts from Current Literature

Ear

PROTEIN-BODY THERAPY IN CHRONIC ECZEMA OF THE EAR. HERMANN STERNBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:200 (Jan. 31) 1927.

A nonspecific milk preparation, autoserum and autovaccine were used without any other therapy. Secondary infection, swelling and furunculosis disappeared. In only one patient with furunculosis did the milk preparation fail to give any results. Autoserum was also ineffective, but autovaccine cured the patient, although the eczema was not affected. The results of injection of protein bodies were negative for eczema. The treatment is highly recommended for secondary furuncles.

ACUTE ABSCESS OF THE TEMPORAL LOBE AFTER ACUTE OTITIS MEDIA. PAUL SAUER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:202 (Jan. 31) 1927.

A girl, aged 12, had suffered from pain in the right ear for seven weeks before she was admitted to the hospital. There was a discharge from the ear for several days before July 12, 1925, when the patient entered the clinic. Two days previously she had had fever and vomited. The temperature was 102.2 F.; the pulse rate, 128. After the child had been in the hospital for six days, a high temperature suddenly developed, with severe headache, a feeling of fatigue, vomiting and stiffness of the neck. A radical mastoid operation was performed on July 19, by Professor Preysing. The tympanum and antrum were surrounded by bone as hard as ivory and contained pus and granulations. On account of the presence of meningitis (stiffness of the neck, a positive Kernig sign, cloudy spinal fluid with numerous leukocytes), the dura of the middle and posterior cranial fossae was exposed, and an extradural abscess was found in the middle cranial fossa.

The patient was referred to the outpatient department on August 22 and readmitted on August 25, on account of severe headache, vomiting and fatigue. There were no meningeal symptoms and no nystagmus. Choked disks with hemorrhages into the retina were present. On September 12, the patient lost control of the sphincter. On September 19, the symptoms had increased. The choked disks became plainer (4 diopters on both sides); this was more pronounced on the left side. There was almost complete hemianopsia on the left side. Optomotor nystagmus (Cords) was absent to the left. Visual radiation was missing in the right eye. The observations of the eye proved the existence of an abscess of the right temporal lobe. Professor Preysing slowly evacuated 250 cc. of thin pus. A bent probe protected with moist cotton did not encounter any capsule. On Jan. 9, 1926, the patient was discharged well. A bacteriologic examination was not made.

In the great majority of cases, otitic abscesses of the brain are not caused by diseases of the mucous membrane in the cavities of the temporal bone, but by the diseased bone which almost invariably reaches to the dura. Experienced observers have reported forty cases of otitic abscess of the brain, in thirty-seven of which the bone was diseased. In one case the diseased bone did not reach to the dura, and in only two cases was it healthy. Manasse's "Acute Osteomyelitis of the Anterior Portion of the Skull in Acute Suppuration of the

Accessory Cavities" illustrates well the path of infection in the class to which the case reported belongs, when he says: "The most pronounced microscopic changes were found in the macroscopically intact bone. All vessel channels were so tightly filled with pus cells that the vessels could scarcely be recognized as such."

INFLUENCE OF VISION ON THE VESTIBULAR TURNING AND AFTER-NYSTAGMUS: SECOND COMMUNICATION. J. OHM, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:259 (Jan. 31) 1927.

1. In the turning experiment in the dark a purely vestibular nystagmus occurs in jerks in the direction of turning. It is followed by a strong after-nystgamus of long duration, in the opposite direction.

2. In the light, with uncovered eyes, pendulum-like nystagmus occurs which is of greater amplitude and frequency. It is a mixed nystagmus of an opticovestibulary nature, which the author named an opticovestibulary turning nystagmus. The term "railroad nystagmus" (Bárány) is supplanted by the term "optic turning nystagmus." Scarcely any after-nystagmus follows when the conditions of the experiments of the author are observed. The state of excitation caused by turning is almost instantaneously suppressed by vision.

3. If strong convex or frosted glasses are placed over the eyes, an intermediate stage between the nystagmus in the light and in the dark is found, and the after-nystagmus becomes stronger. The vestibular component is more pronounced when frosted glasses are used instead of strongly convex ones. In one instance psychic interferences also seemed to be present.

CONTRIBUTIONS TO THE CLINICAL SIGNS AND HISTOLOGY OF MALIGNANT TUMORS OF THE TEMPORAL BONE. C. RUF, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**: 274 (Jan. 31) 1927.

Malignant tumors of the temporal bone which involve the organ of hearing may be primary tumors or tumors in the surrounding tissue which have penetrated into the temporal bone. Primary and secondary tumors of this nature are recognized by abolition or decrease in function of the acoustic and static organs, which occurring simultaneously with other clinical symptoms, make exact localization possible. The microscopic picture frequently shows a peculiarity, as tumors are observed which occur rarely in the organ of hearing or elsewhere.

Four cases are reported in detail: (1) A case of myxofibrochondroendotheliosarcoma of the left temporal and sphenoidal bone, in which the patient, a man, aged 49, was treated with the roentgen rays following a diagnosis of tumor of the brain. (2) A case of spindle cell sarcoma emanating from the posterior wall of the right osseous meatus in a man, aged 45. Surgical removal was followed by treatment with radium and the roentgen rays. The patient was living three months after the operation, without a recurrence of the tumor. (3) A case of endothelioma of the sheath of the acoustic nerve with extension to the left temporal bone, in a woman, aged 60. The mucoperiosteum of the middle ear was intact. The tumor originated from the middle cranial fossa and penetrated into the recessus epitympanicus and the other middle ear cavities after destroying the osseous tegmen tympani. (4) A case of primary squamous epithelium carcinoma of the outer meatus which penetrated into the cavities of the middle ear and thence into the temporal bone. The patient, a woman, aged 49, underwent an operation on May 8, 1925, and died on Jan. 22, 1926, of marantic atrophy.

HISTOLOGIC AND CLINICAL INVESTIGATIONS CONCERNING THE CRITICAL EXAMINATION OF THE INDICATION FOR OPERATION IN ACUTE MASTOIDITIS. OTTO MAYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:301 (Feb. 22) 1927.

Histologic observations have shown that a severe inflammation of the mastoid process was found in patients who have undergone operations on the fourth and fifth days of the course of acute mastoiditis. Suppuration and purulent hemorrhagic exudation into the covering of the pneumatic spaces and into the cavities themselves is preeminent. The mucous membrane is mostly preserved, but greatly thickened on account of edema, infiltration with leukocytes and enormous dilatation of the blood vessels. The infiltration of the mucous membrane may turn into suppuration. Hemorrhages in the tissue and thrombosis of the vessels are noticed. The blood vessels in the vessel channels of the bone are greatly dilated. Here also perivascular infiltration and occasionally osteoclasts are found, mostly in connection with the wall of blood vessels and in deep lacunae of the bone.

New formation of bone is also visible at this stage in the form of small osteoid edges on the bone at the walls of pneumatic cells, rarely at the walls of vessel channels. In patients who underwent operations on the tenth day and later, the pathologic changes in the bone had advanced. Practically the same changes were found, but the mucous membrane was changed into granulation tissue almost everywhere. A decided change could be noticed after the eighteenth day. An extensive rarefaction of the bone was present. This could be found in preexisting vessel channels, but it was more extensive in those newly formed. The bone was destroyed, and large cavities had been created, which were filled with granulation tissue. Perforations develop in the same manner, namely, through resorption of bone in newly formed channels (vascular ostitis), and not through arrosion of the wall of the pneumatic cells (Krainz). In the further course, fibrous tissue is formed at the border of the spaces created by resorption. This osteoblastic marrow is the origin of a new bone formation, the nature of which is determined by the type of periosteum. Inside this layer is granulation tissue which surrounds the suppurating cavity. The same arrangement of layers (peripherally, fibrous marrow and new bone; centrally, granulation tissue) is found in the cells. Just as frequently cells are filled with fibrous tissue. In this tissue the formation of new bone occurs either at the border in the form of osteoid aggregations or in the form of trabeculae, or both forms are found simultaneously.

The formation of new bone is independent of perforation (Krainz). It begins during the first days of the disease (Manasse) in the form of osteoid edges, but it reaches greater dimensions only in the fourth week. It is dependent on the formation of fibrous marrow. Although osteoclasis always occurred, the author observed in two patients that the preponderance of formation of new bone occurred in the eighth week.

A complete cure is possible, but the course of the disease is not definitely marked. Healing takes place on the surface, and the process continues underneath. Acute exacerbations frequently occur. Only in the beginning of the disease, when the cells are filled with pus, is the term "empyema" in order. Later, a collection of pus is present in a granulating cavity, that is, an abscess. It is important to note that even in the beginning of the disease a severe suppurative inflammation can exist in the mastoid process which is essentially localized in the perivascular infiltration, and osteoclasts can be found in the vessel space. Pronounced rarefaction and macroscopically visible disease of the bone are not found, however, until the fourth week of the disease.

Clinical observations in thirty patients showed that most complications occur before a macroscopic disease of the bone is observed. The complications occur through propagation of the inflammation through phlebitis and periphlebitis from the numerous vessels of the mucous membrane and the bone of the inflamed mastoid process (osteophlebitis-Koerner). The course of the inflammation is dependent on the species and virulence of the microbes. The epidemicity plays a great part, as the frequency of grave cases in the beginning of 1926 showed. The constitution of the patient and the pneumatization of the mastoid process are important.

Clinical symptoms should determine the indication for operation. Operation should always be performed when it is probable that there may be suppurative inflammation of the mastoid process and dangerous complications will occur in case of delay. It is useless and dangerous to wait until symptoms of a disease of the bone or a perforation occur. A "late operation" becomes frequently a "too late operation," because the disease has advanced to parts where it cannot be reached or because it leads to a fatal general infection, or to intracranial complication. One cannot wait for a positive roentgenogram. The clinical symptoms alone are sufficient. A late operation does not protect the patient from a secondary operation. It also requires a longer time to heal. If a conscientious consideration leads to the indication of an early operation, it must be performed in order to prevent a fatal outcome in foudroyant cases. It is technically more difficult than most late operations, but the fever disappears sooner, complications may not be expected and the chances of healing are the best.

The conception of "mastoiditis" must be formulated anew. Not only diseases of the bone of the mastoid process (ostitis, osteomyelitis, endostitis) must be diagnosed mastoiditis, but all diseases of the mastoid process which are protective measures against injuries to the body, be they alterative, exudative or proliferating. The general conception is erroneous that mastoiditis is identical with ostitis. It is falsely assumed that only the disease of the bone is dangerous because it leads to perforations. In fact, fatal complications set in frequently at the time when the bone is only slightly diseased, and when it is impossible to recognize a disease of the bone macroscopically or by the roentgen rays. Therefore, it is not always correct to wait for signs of a disease of the bone, because then operation may be too late.

AMBERG, Detroit.

Pharynx

THE INTRACUTANEOUS REACTION WITH STREPTOCOCCUS HEMOLYTICUS IN HEALTHY PERSONS AND IN PATIENTS INFECTED WITH THE STREPTOCOCCUS, WITH ESPECIAL CONSIDERATION OF THE TONSIL. ERICH WIRTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:245 (Jan. 31) 1927.

The intracutaneous injection of an emulsion of hemolytic streptococci (killed by heating to 140 F. for fifteen minutes) causes the same reaction in healthy and in diseased persons. The degree of reaction is the same when the stocks used are taken from pus, erysipelas or scarlet fever, either virulent or weak. The infecting stock does not provoke a specific reaction in the organism infected by it. Therefore the intracutaneous reaction with streptococci from the tonsil does not allow one to draw conclusions concerning the pathogenic importance of these germs for any general disease. The skin reacts differently in different persons. The possibility of reaction seems to be small in a serious infection.

No relation was found between the reaction of the Dick toxin and the intracutaneous reaction with streptococcus vaccine, nor between the Dick reaction and the course of the disease.

AMBERG, Detroit.

Nose

OPHTHALMIC FINDINGS IN NASAL ACCESSORY SINUS DISEASES. CYRIL BARNERT, New York State J. Med. **26**:1017 (Dec. 15) 1926.

The author states that it is well known that the eye may be involved in disease of the adjacent nasal cavities, but that the true significance and importance of these conditions in the eye as a diagnostic aid are perhaps not fully appreciated. He points out briefly the more striking and illustrative facts elicited by routine examination, paralleling his report with the published reports of other observers in the past year or two.

The external eye, the lids, conjunctiva, musculature, episclera and sclera rarely show abnormalities in nasal disease, except that local infection in the sinuses is occasionally expressed in conjunctival injection or as an episcleritis or scleritis, or when an accompanying periostitis of the orbit in ethmoiditis produces an edema of the lids with proptosis or bulging of the eyeball, together with impaired motility of the globe.

He reports a case of simple edema of the orbit clearing up under conservative intranasal treatment.

He also reports a case of orbital abscess in an infant 10 days old, with autopsy showing generalized sepsis with abscess of the right antrum which had perforated into the orbit.

Another orbital abscess was in a boy, aged 11, who fell ten feet two days previously. The patient was unconscious for ten minutes. The next day there was intense edema of the left eyelid. The temperature was 102 F. Tentative diagnosis was fracture of the base of the skull through the left ethmoid with beginning orbital abscess. Pus was aspirated from the upper inner quadrant of the orbit, and the abscess was opened and drained. The author reopened the orbit, exposed the lamina paparacea, demonstrated a fissure fracture therein, and proceeded to exenterate the ethmoid from the orbital side. Recovery was uneventful.

The author states that iritis, uveitis and chorioretinitis are comparatively rare as sequelae of sinusitis, but are occasionally found. He believes that it is important to eliminate nasal causes of such conditions, especially when operation on the eye is contemplated.

The author states that papilledema is occasionally found as a complication of focal infection in accessory sinusitis.

The question of retrobulbar optic neuritis is of the greatest importance for two reasons: first, because of its frequency; and second, because of the absence of any objective evidence in the eye to suggest its presence.

Clinically, retrobulbar neuritis occurs in acute and chronic form. The acute type manifests itself by headache, a rapidly progressive loss of central vision, together with concentric contraction of the peripheral fields, first for color and then for form, also tenderness on pressure on or movement of the eyeball. In the chronic form, the symptoms are less acute, with a dull ache or no pain and concentric contraction of the fields with or without central scotoma.

He reports two cases of the acute type and four cases of the chronic type cured by operation and one case of the chronic type not cured.

Adding his cases to those found in the published reports of the last two years, the author brings out the following facts: There was a total of 124 cases of retrobulbar neuritis. Those due to endonasal causes were fifty-three, or over 40 per cent; and in all these cases, when any mention of therapeusis was made, recovery followed intranasal intervention. Forty cases were attributed to multiple sclerosis, and five cases to intracranial tumors, including an aneurism of the circle of Willis, a hypophyseal cyst, a suprasellar tumor and two frontal lobe neoplasms. Three cases were ascribed to malaria; three to toxemia, one of these metastatic, one caused by wood alcohol and one by arsenic, and a series of fifteen cases with spontaneous recovery was reported.

In concluding, Barnert states:

1. No examination of the eye is complete without a careful plotting of the visual fields.

2. In the presence of ocular inflammation a thorough search for the underlying cause must be made, and the cause removed if possible. In all cases of retrobulbar neuritis, optic neuritis and papilledema, a complete neurologic examination is indicated in addition.

3. In all cases of retrobulbar neuritis in which no other cause can be found, the ethmoids and sphenoids should be exenterated, even if there is no clinical or roentgenologic evidence of disease of the sinus.

MUCOCELE OF THE FRONTAL ETHMOID WITH EXTENSIVE INVASION OF THE ORBIT. L. REVERCHON ET TSIROS, Arch. internat. de laryngol., 1926, p. 165 (translation from the abstract by Rene Gaillard, Ann. d. mal. de l'oreille, du larynx **46**:95 [Jan.] 1927).

The authors present a case of a young sailor with a tumor at the internal angle of the left orbit. The tumor was the size of a walnut, and shoved the eye out, producing a lowering of vision by crystalline opacities and a certain degree of papilla stases. This tumor had grown for ten years at first slightly, and more rapidly recently. No anterior trauma and no intranasal symptomatology were present.

At operation a large bony gap was found occupying the floor of the frontal sinus and extending back to the luxated lacrymal bone. This pouch contained a mucopurulent liquid of the color of coffee with cream in it. No osteitis was present. The mucosa which covered the cavity was removed in a mass. There was no trace of the nasal frontal canals. Healing took place by primary intention of the operative wound, which was sutured without nasal drainage. The eye took its place, but the crystalline opacity persisted.

The authors call attention to the extension of the cystic tumor far back into the orbit and to the presence of disturbances of the trophic lens by compression of the ophthalmic ganglion. This mucocele was comparable to an infected paradental cyst.

SARCOMA OF THE NASOPHARYNX IN A CHILD WITH INTRACRANIAL INVASION. BRINDEL, RETROUVEY, GUINAUDEAU, Rev. de laryngol. **3**:101 (Feb. 15) 1927.

Malignant tumors of the nasopharynx are rare in adults but exceptional in the infants. In 70,000 patients examined in the Clinic at Bordeau in twenty years, thirty-four were observed; only three occurred in patients under 30 years of age. A case was reported in a boy, aged 7, who had a sarcoma of the nasopharynx and was under observation in the children's ward for six months. The mother was also in the clinic for a disarticulation of the hip because of sarcoma of the femur.

Autopsy showed that both hemispheres were intact. A lobulated tumor about the size of a goose egg occupied the center of the base of the cranium. There was a prolongation into the left orbit. The right orbital cavity was entirely filled. The hypophysis was invaded, and there was involvement of both anterior and posterior clinoid processes. There was invasion of the nasal fossae, ethmoid and sphenoid. The pathologic condition was that of fibrosarcoma.

The literature does not have much bearing on the heredity of malignancy. The history in the present case showed both mother and child in the hospital at the same time, and both had sarcoma.

Irradiation with the roentgen ray was tolerated well except for the initial shock. The bones of the cranium offered resistance to the rays, and could the tumor have been exercised as in the larynx, it is possible that the radiations may have accomplished more promising results.

ROTH, Kankakee, Ill.

ORBITAL CELLULITIS OF NASAL ORIGIN AND INFLAMMATORY ETHMOIDITIS. DELAGE, Rev. de laryngol., April 30, 1926 (translated from the abstract by Seigneurin, Ann. d. mal. de l'oreille, du Larynx **46**:93 [Jan.] 1927).

The reactions of the cellular tissues of the orbit following inflammation of the adjacent cavities have already been made the object of numerous studies, but in 1921 Vernieuwe described a particular form of these reactions which accompany certain types of ethmoiditis: inflammatory ethmoiditis which has been individualized.

Delage does not believe that orbital cellulitis occurs only in the ethmoid. All the sinuses that encircle the orbit can cause these same reactions. However, the ethmoid has more constant and more important relations with the orbit. The orbital swelling is only an inflammatory reaction and an expression of the infectious processes that extend to it. Only a prephlegmon would be stabilized at one moment of its evolution. Why this stabilization? One can only make a hypothesis based on the defence of the soil and on the strength of the attack (microbic virulence). Such reactions are peculiar in that they reappear easily. The tissues keep a special sensibility, so that there is a frequency of crises, even when the ethmoiditis is chronic. Diagnostic precision is not always easy. All means of exploration should be used.

VAIL, Cincinnati.

EMPYEMA OF THE FRONTAL SINUS CAUSED BY THE STREPTOCOCCUS MUCOSUS. LUDWIG PESTI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:192 (Jan. 31) 1927.

A woman, aged 23, was admitted to the hospital on Jan. 22, 1926, with the history that she had suffered four weeks previously from violent headache following a coryza. The headache became less, even ceased, but reappeared. For a week before she came to the hospital the left side of the forehead had been swollen, and the skin had become red and painful. An incision revealed a perforation in the outer wall of the frontal sinus. The purulent discharge contained *Streptococcus mucosus* in pure culture. Professor Paunz performed Killian's operation on Jan. 28, 1926. In three weeks the patient was cured. A mucosus sinusitis may be present when there are few or no changes in the nose, but disease of the bone sets in early (periostitis, subperiosteal abscess, with meningeal and cerebral symptoms). An early diagnosis is important. Neither the clinical picture nor the roentgenogram indicates the degree and extent of the destruction of the bone. Therefore, only an early radical operation prevents serious and often fatal complications.

AMBERG, Detroit.

Miscellaneous

A Further Communication Concerning the Local Diagnostic Value of the Wassermann Reaction in the Spinal Fluid, Especially Concerning the Differential Diagnosis of Difficult Cases of Syphilis or General Inflammatory Diseases in the Cranium. Johannes Zange, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:234 (Jan. 31) 1927.

In a previous communication the author stated that the Wassermann reaction in the spinal fluid was of importance in differential diagnosis in certain cases, and that this circumstance applied to cases which clinically resembled syphilis of the central nervous system and its membranes. If the patient had an old case of syphilis with a positive Wassermann reaction of the blood, the difficulty of a differential diagnosis is obvious. In these instances the Wassermann reaction of the spinal fluid could be a deciding factor if the reaction was caused by the spinal fluid itself and not by the blood from which the reagent had entered the spinal fluid by virtue of the increased permeability of the meninges.

The whole question of the value of the Wassermann test in general meningitis, with diffuse as well as with circumscribed permeability, and in abscess of the brain, with a locally defined meningitis, has not been cleared up sufficiently. It may be assumed that a Wassermann reaction may be used as a local diagnostic means and may prove the presence of syphilis of the central nervous system, if a positive Wassermann reaction is present in the spinal fluid as well as in the blood. It may be assumed that the active substances in the spinal fluid did not migrate from the blood if the total albumin is found only slightly increased. If, in such instances, the Wassermann reaction of the blood is only weakly positive, this conclusion seems to be still more justified.

The author cites two cases in point. The first patient, a man, aged 52, suffered from an acute miliary tuberculous meningitis and syphilis (old syphilis of the tongue and a mesaortitis syphilitica not yet quiescent), with a positive Wassermann reaction in the blood and a negative Wassermann reaction in the spinal fluid. The second patient showed the deciding part of the Wassermann reaction in the spinal fluid, especially if the colloidal gold reaction fails. This patient, a man, aged 36, apparently suffered from a slow, traumatic, late meningitis after a fracture of the base of the skull with a complicating infection of the middle ear. A major surgical operation seemed to be indicated. The positive Wassermann reaction in the blood and in the spinal fluid indicated another procedure. Only the granulations in the outer meatus were removed. Antisyphilitic treatment was successful.

Kraemers, Eicke, Zadek, Lesser and Stern have reported a positive Wassermann reaction in the spinal fluid in epidemic or suppurative or tuberculous meningitis (partly found in necropsies) when syphilis was undoubtedly not present and when the Wassermann reaction in the blood was negative. F. Plaut (mentioned by Kafka) claims that a poor technic must usually be blamed in these instances. Zaloziecki has paid attention to this question for years, and he has never seen a positive Wassermann reaction in the spinal fluid in the absence of syphilis. A case of Zaloziecki is cited. The patient, a prostitute, showed a strongly positive Wassermann reaction in the blood. She suffered from general tuberculous meningitis with pronounced pleocytosis (half polymorphonucleosis) and a total albumin content of 0.2 per cent.

Three successive spinal punctures gave a negative Wassermann reaction of the spinal fluid. Zaloziecki quotes this case to show that in cases of a clear meningitis, with a positive Wassermann reaction in the blood and in the spinal fluid, the conclusion could not be drawn that the greater permeability in meningitis would have allowed substances which cause a positive Wassermann reaction to enter the spinal fluid from the blood.

BASAL CEPHALOCELES. WILHELM SCHREYER and WALTER SPRENGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:252 (Jan. 31) 1927.

The authors observed a patient who exhibited a cephalocele in the meatus in the squamous portion of the temporal bone and in the orbit. The patient, a man, aged 37, at birth showed a protrusion of the right eyeball, swelling of the right upper lid and an elevation in the right temporal region. Nothing was known of a trauma at birth. The right eye showed a dystrophy of the cornea, old maculae and some fresh infiltrations, besides a great decrease of sensibility. Examination of the interior of the eye was impossible. There was minimal vision. The right upper lid was thickened. The lacrimal gland was protruding. The eyeball was dislocated. It was 18 mm. deeper in the orbit than the left corresponding eyeball, and the center of the cornea approached the midline 9 mm. nearer than did that of the left eye. All motions of the bulbus were interfered with. The exophthalmos reached about 20 mm. The pulsation of the bulbus was 2 mm. Palpation revealed an elastic mass in the orbit. The eyeball could be pushed back for 2 cm. without causing symptoms in the brain. Compression of the common carotid did not affect the exophthalmos nor the pulsation. No fremitus and no synchronous pulsations could be detected such as are found in genuine exophthalmus pulsans. The squamous portions of the right temporal bone showed a defect in the bone about the size of a dollar, with smooth edges. Pulsation could be felt plainly. At the upper and anterior wall of the right osseous meatus a protrusion the size of a small pea could be seen immediately in front of the membrane of the drum. It covered the anterior upper quadrant of the membrane and the anterior half of Sharpnell's membrane. The membrane of the drum was normal. The protrusion showed slight pulsation and could easily be pushed back with a probe. It was tense, grayish pink and appeared to be covered with a thin layer of epidermis. The whispered voice could be heard in both ears for a distance of 12 m.

Stadtfeldt collected ten cases of this nature from the literature in 1902. In two necropsies, large defects of the ethmoid bone were present. In a case of Oettinger the whole roof of the ethmoid, to the crista galli, the largest part of the orbital plane of the frontal bone and the small wing of the sphenoid were missing. In Tauber's case the whole cribriform plate and portions of the sphenoid bone and orbital walls were absent. Three large defects also existed in the cranial bone.

A wrong diagnosis may be of disastrous consequences as a case of Pilpel proves. A diagnosis of adenoid vegetations was made, and adenotomy was performed. Necropsy showed a large oval opening (2 by 1.5 cm.) in the sphenoid with protrusion of the brain and hypophyseal tissue. This condition is most frequently found in nurslings who cannot live. Exner collected six cases from the literature and recommended an operation similar to an operation on the hypophysis, because these tumors, he claims, are exposed to an early oral infection. The authors quote the literature concerning the various other forms of cephaloceles, and refer especially to the work of Stadtfeldt.

AFTER-TREATMENT OF OTOGENOUS ABSCESS OF THE BRAIN WITH RIGID CANNULAS. O. KOERNER, Ztschr. f. Hals-, Nasen.- u. Ohrenh. **17**:293 (Jan. 31) 1927.

From the time of the introduction of the cannula in June, 1921, until December, 1926, the author observed six cases of abscess of the cerebrum and four cases of abscess of the cerebellum. Three patients with cerebral abscesses without complication and the four with cerebellar abscesses were cured. Of the other three patients with cerebral abscesses, two suffered also from meningitis, which was diagnosed before the operation. In one case the condition was not recognized because the patient entered the clinic in dying condition, suffering from a carcinoma which had penetrated into the mastoid process. Necropsy showed that the suppurating tumor had penetrated into the right temporal lobe, and had produced an abscess the size of a pigeon's egg. Although the good results in these few cases might be a lucky coincidence, it might be worth while to employ and test the method of after-treatment used by the author. In the after-treatment, no packing but a rigid cannula is used. The cannula is slightly bent, but straight cannulas can be employed when the abscess is opened from the cranial wall and not from the diseased temporal bone. The end of the cannula has oval-shaped openings at the side in addition to the central opening.

The author has published his method of after-treatment in the fifth edition of "The Otitic Diseases of the Brain, Meninges and Blood Vessels," which he wrote in collaboration with K. Gruenberg, published by J. T. Bergmann, of Munich, in 1925.

AMBERG, Detroit.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, Feb. 7, 1927

ARTHUR M. CORWIN, M.D., *President, in the Chair*

PRIMARY TUBERCULOSIS OF THE SINUSES. Presented by DR. JOSEPH C. BECK.

A negro, aged 17, admitted to the Research and Educational Hospitals of the University of Illinois in July, 1926, with evidence of interference with vision, did not return until seven months later when he had lost a great deal of vision. The eyes protruded, he had pressure symptoms and gave a history of having had nasal hemorrhages. Sarcoma was suspected. The Wassermann reaction was negative, and there were no symptoms or signs of syphilis; examination of the chest was negative. Radical surgical treatment was decided on. Part of the middle turbinate body was removed for biopsy, and examination revealed tuberculosis which had extended through the frontal bone and into the other structures. Shortly afterward, Drs. Lederer and Livingston performed a radical operation by incision over both eyes. Broken-down masses of granulation tissue and caseous material were found. The dura was thickened and covered with caseouslike granulation tissue and was not interfered with. The wound was closed and drainage established intranasally and externally. The patient made an uneventful recovery, and there had been some improvement in vision, in ocular perception and in recognition of objects. The pus obtained from the region of the frontal sinus was sterile, but on a later growth staphylococci were found. No tubercle bacilli had been recovered, and animal inoculations had not yet been completed.

Examination of the eyes showed swelling of the nerves and later a choked disk, but there was no change in the eyeball. The characteristic facial changes were pronounced, owing to the breaking down of the supra-orbital bones, and because of infiltration of the rectal muscles, the patient was not able to hold the eye in proper position.

Roentgenograms and photomicrographs were presented showing the typical giant cells and tubercle formation.

CARCINOMA OF THE LARYNX. Presented by DR. SAMUEL SALINGER.

A man, aged 56, first seen in November, 1926, was suffering with considerable dyspnea due to a neoplasm which occupied the greater part of his larynx. Examination disclosed a rather uniform infiltration in the region of both ventricular bands, which seemed to extend down deeply into the larynx. A specimen was removed and biopsy showed an infected epidermoid carcinoma.

Laryngectomy was performed on Dec. 10, 1926, under scopolamine, morphine and local anesthesia, without any general anesthetic. There was extensive infiltration of the larynx and perilaryngeal tissues. On exposing the larynx, the thyroid isthmus was found attached to the larynx and infiltrated, the sternohyoid muscle on the right was involved, and several lymphatic glands seemed to be involved. The prognosis seemed so unfavorable that extensive operation appeared to be useless, but nevertheless, all the affected tissues were excised. There were adhesions due to a previous tracheotomy. The technic brought out by MacKenty

was used. The patient was fed through an esophageal tube for four weeks. Then the tube was removed, and it was found that there was a passage large enough to permit the swallowing of food. He has eaten without difficulty since that time and has gained 25 pounds (11.3 Kg.) in weight.

Histologic examination of the specimen removed showed a typical squamous cell epithelioma.

DISCUSSION

DR. BECK: The after-care of such patients is the most important factor in connection with laryngectomy. The operation itself is not difficult after one has mastered the points emphasized by MacKenty. Drainage after the operation to avoid infection is of great importance.

It is time that the roentgen ray is given its proper valuation, and physicians should state that they have never seen a case in which carcinoma remained cured after roentgen-ray therapy. Laryngectomy gives such patients a chance for permanent cure. If the patient is promised that he will be able to speak following laryngectomy, that the operation will save his life, and that he can be educated to talk by means of an artificial larynx, the operation will not seem so formidable.

Tracheal fistulas often occur even after careful operations, but this is not an indication against the operation. The condition of the glands of the neck should be noted. Cases have been reported in which the infection has disappeared following laryngectomy and removal of the glands. The presence of infected glands is not a contraindication for the operation.

DR. ALFRED LEWY: Dr MacKenty has worked out the after-treatment in great detail. He told me that he had never seen a patient recover from an extrinsic laryngeal carcinoma.

DR. SALINGER: Dr. MacKenty is very particular about the patients he selects for laryngectomy, but other surgeons have performed laryngectomies and removed infiltrated thyroids and glands and the patients have recovered. Some patients have enlarged glands which are not truly carcinomatous. The glands should always be sectioned and examined carefully, for sometimes inflammatory conditions cause the same symptoms. The after-care is very important. My patient was a Slovenian who could not speak English or German and could not write or read any language but his own, so it was difficult to give him instructions in speech.

PAIN IN THROAT DUE TO FOREIGN BODIES. Presented by DR. BECK.

The patient, referred to Dr. Beck because of a pain in the throat, said that an attempt had been made to remove some clips which were applied following tonsillectomy performed in another city. A roentgenogram showed six of the clips in the throat. Dr. Beck had attempted to remove the clips under the fluoroscope but was unsuccessful. By direct illumination, he was able to remove four of them, but two still remained in place, although the patient was unaware of the fact.

This case was reported to demonstrate that the presence of a foreign body in the throat would cause bitter complaint if the patient knew it was there, but that there would be no complaint if the patient was unaware of its presence. Dr. Beck believed that the presence of the clips had nothing to do with the pain of which the patient complained.

DISCUSSION

DR. LOUIS OSTROM, Rock Island, Ill.: I have used clips for many years, and during the last fifteen years I have had to visit a patient following tonsillectomy

on only six occasions. I have performed many hundreds of tonsillectomies and probably 400 radical operations for quinsy. I have never had to use a ligature. After the tonsils are removed, the hemostat is used. If that does not stop the hemorrhage within a reasonable time, I use Stockton's needles for suturing the tissues. If the tissue is too solid to permit the use of this suture without causing bleeding, I apply the clips, making a circle around the blood vessel. In this way I have no difficulty in controlling the hemorrhage, and I have never been called back to take care of hemorrhage within the twenty-four hours following the operation. If the bleeding persists, I sew in a pad. I use the clips only when necessary, but I do not know of any patient who is carrying the clips who has had any discomfort. I try to take them out as soon after operation as possible, but I explain to the patient that they can be removed at any time. It is safe to use them when necessary.

Dr. W. E. Grove, Milwaukee: I have recently seen a patient in Milwaukee who had been operated on by someone else several years prior to presentation. A local tonsillectomy had been done. A needle had been left in the pharyngeal tissue, but the patient had not experienced any trouble.

Dr. Robert Good: It is dangerous to use such clips for fear that they may drop off and get into the larynx, or be aspirated into the bronchi.

Dr. Beck: I am glad to know that the foreign bodies do no harm if permitted to remain in the larynx.

The Relation of the Oral Articulative Movements of Speech and of the Extrinsic Laryngeal Musculature in General to Vocal Cord Function. Presented by Dr. Elmer L. Kenyon.

The larynx, hyoid bone, lower jaw, tongue, soft palate and pharynx, together with their combined intrinsic and extrinsic laryngeal musculature, constitute one united system of organs and muscles for all functions that involve the larynx. Their functions are primarily those of animal life; secondarily, in man, they are employed also as organs of speech. The intrinsic and extrinsic musculature can act only as one united muscular system whether for animal functions or for speech. No other psychoneurologic means of action is provided. The peripheral physiology of speech is immediately based on the corresponding more basal physiology of the animal functions of the same organs. That the psychology of the peripheral speech function is based also largely on that of certain corresponding animal functions of these organs, is probably true. The extrinsic musculature is attached to the larynx almost exclusively by way of the thyroid cartilage, and thus may apply its laryngeal influence directly to the vocal cords, and, through cooperation of the thyroarytenoid muscles, presumably to altering the details of vocal cord approximation. Studies of laryngeal function, apart from observation within the larynx itself, have been carried out on a wrong principle. Attempt to understand the action of so complex and intelligently cooperative a musculature by first studying the anatomy and then speculating as to how the muscles probably act, leads to inadequacy of result, as well as to error. It is possible to know the actual performance of such a musculature only by observation of what it actually accomplishes. Study by the author of the extrinsic musculature in actual action, by palpation of the upper anterior tip of the thyroid cartilage and adjacent structure, including the trachea, has yielded important physiologic information, both as to normal and to abnormal speech. One important error in physiologic observation, that regarding the manner in which the vocal cords are stretched for purposes of elevating the pitch of the voice, has pervaded the textbooks of

laryngology for at least seventy-five years. Actual observation, by palpation, of the action of the thyroid cartilage, shows clearly and definitely that elevation of the vocal pitch is produced not by tilting the thyroid cartilage downward, as has been supposed, but by tilting it upward. This is an act of the extrinsic musculature. By similar observation of the actual action of the extrinsic muscles, the whispered voice is found to be produced by joint action of the entire intrinsic and extrinsic musculature, but with certain important differences from their action in the production of the loud voice. Cerebral excitation for the loud, as compared with that for the whispered, voice may possibly differ not merely in degree, but also in kind. The interrelation between the movements of the organs of the mouth that determine the elemental sounds of speech, and the approximation of the vocal cords for the production of voice, are so immediately related as to constitute one complex physiologic act.

DISCUSSION

Dr. Good: Prof. M. Metfessel, of the University of Iowa, Iowa City, is studying the singing voice. He takes moving pictures of the vocal organs during singing, and in the pictures the slightest flatting or sharping of the voice is indicated. In addition to taking the moving picture of the vocal organs, he takes a moving picture of the person who is singing and makes a record of the singing voice on a wire. In these three ways, he analyzes the singing voice. The picture is taken by means of a small electric light and a small mirror. As the patient sings, the vibrations of the breath make the mirror vibrate and the picture of the reflected light is shown on the screen. I have visited his laboratory and have seen the work. The wire is charged and runs from one spool to another and will keep the record of the voice for hundreds of years. Dr. Metfessel asked what formed the vibrato and where it was. I thought that it was in the diaphragm and asked him to make moving pictures of the abdomen, but he thought this was not the proper method of determining when the vibrato is made. I wish to ask Dr. Kenyon if he has any idea as to whether it originates in the larynx, in the diaphragm, or in both.

Dr. Lewy: I have observed on my own larynx that as the pitch goes higher, not only the hyoid, but also the cricoid, moves upward, and the interspace in front becomes smaller as the pitch rises. As I understand Dr. Kenyon, this is not in accordance with his observation.

Dr. Beck: Will Dr. Kenyon explain the whisper of the laryngectomized patient, and also, whether he has observed or made use of the nasopharyngoscope and the Hays pharyngoscope in watching the movement of the cords during whispering?

Dr. Robert Sonnenschein: So far as I am aware, no one has done such valuable physiologic work on the voice as Dr. Kenyon.

Does the cricothyroid muscle act as a tensor of the cords? It is unique in that it is supplied by the superior laryngeal nerve, all the others being supplied by the inferior or recurrent laryngeal nerve.

Concerning Dr. Good's report of the work of Professor Metfessel, I know nothing about the method used by him, but there is nothing new about throwing the voice on the screen. It was done many years ago by Dayton C. Miller of Cleveland.

Dr. Kenyon: I am cognizant of the work of Professor Metfessel in Iowa City, but I have no clear knowledge of what they are doing and do not wish to discuss it.

Replying to Dr. Lewy, the physiologic facts are just beginning to be understood, and not much is definitely understood thus far, but in studying the action of the thyroid cartilage in the production of the voice one sometimes finds the thyroid cartilage moving up against the hyoid, and both moving up against the base of the tongue for the higher pitch effects. There is nothing abnormal in Dr. Lewy's observations.

I do not know what the cricothyroid muscles do, but the extrinsic muscles are much more powerful and take hold of the cartilage in a much better way. It seems to be a matter of cooperation of many muscles, and just what the function of the cricothyroid is, I do not know. The old conception of laryngologists is due to the fact that they did not conceive of these muscles as being extrinsic, but that the cricothyroid was the one muscle that could stretch the vocal cord.

I know little about the speech of the laryngectomized person, since I have never had an opportunity to study it in a positive way. Many years ago Dr. Grossman made tests and thought he found that somewhere in the lower part of the pharynx the tissues come together in such a manner that vibration is possible; then by forcing the air out of the stomach there was a sort of basal whispered sound. I have given no thought to the production of the vibrato and do not know how to explain it.

PRIMARY CHRONIC HYPERTROPHIC MASTOIDITIS: CASE REPORT. Presented by DR. HORACE R. LYONS.

A man, aged 57, who was examined on Aug. 30, 1924, had had pulmonary tuberculosis in 1894. In 1912, he had scarlet fever; cholecystectomy had been performed and a ventral hernia repaired. This was followed by infection of the left antrum and empyema. In 1921, the patient had influenza, complicated by marked edema of both lungs. In 1922, following this severe attack of influenza in 1921, the patient had a recurring, severe pain in the left ear. During the severe attacks of otalgia, examination of the left ear revealed no abnormality. There was no redness of the tympanic membrane, no bulging, no fever, no mastoid tenderness on prolonged deep pressure. The first attack of otalgia lasted about forty-eight hours and was severe enough to incapacitate him. After this attack, he had no further trouble for several months, when he again had a similar attack of acute severe otalgia on the left side. Examination by several otologists again revealed no evidence of infection of the middle ear. The tympanic membrane was normal. There was no bulging or redness, no fever and no mastoid tenderness on pressure.

The roentgenogram revealed a few cells in the left mastoid at the level of the antrum and above the knee of the sinus, which showed a slight cloudiness as compared to the other cells in the left mastoid and those in the right mastoid. There was no evidence of breaking down of the cell walls. The physicians who were consulted advised against intervention, but the patient had the sensation of fluid in the middle ear and demanded a paracentesis. The left external canal was cleansed as thoroughly as possible, and a paracentesis was performed. With the aid of slight suction, several drops of clean, thin, colorless fluid came through the opening into the external canal. A culture and smear were taken which proved that the fluid was sterile. The patient was completely relieved of the otalgia. The same symptoms again presented themselves in the summer of 1923. The physicians who were consulted advised against operative intervention. The patient demanded a simple mastoidectomy, which was performed on Sept. 14, 1923.

Operative examination showed that there were several mastoid cells at the level of the mastoid antrum which contained a thin colorless fluid. The fluid

proved to be sterile by several smears and cultures. There was no evidence of breaking-down of any cell walls.

The postoperative course was uneventful, and the patient had complete relief from pain until February, 1924, when he again had severe pain in the left ear, accompanied by deafness, and pain on deep pressure over the upper third of the left mastoid. He again had the sensation of dropping of fluid in his middle ear. This attack lasted about twenty-four hours. In the meantime, the infection of the left antrum continued, and the patient consulted a rhinologist in Boston, who advised and performed a radical external operation on July 7, 1924. As soon as the patient was able, he returned to his home in Colorado. The infection of the antrum completely cleared up, and he had no further difficulty until September, 1924, when he again had an attack of severe left otalgia, at which time he came under my care.

On examination, Aug. 30, 1924, I found the tonsils clearly removed; the nasopharynx was normal. The nose showed the septum practically straight; no pus was found. There was no opening into the left antrum in the inferior meatus. There was a scar on the left side above the alveolar ridge, evidently the incision made for the radical operation on the external antrum. Transillumination showed that the light reflex from the left antrum was the same as that of the right.

The tympanic membrane in the right ear was retracted and slightly thickened; otherwise it was normal. The tympanic membrane in the left ear showed several linear scars, apparently the effect of the repeated paracenteses. The hearing tests with tuning forks showed no difference in the acuity in the two ears. A scar was found over the left mastoid, giving evidence of a previous mastoidectomy. The patient complained of pain over a small area at the level of the mastoid antrum on deep pressure.

A roentgenogram taken August 30 revealed a shadow involving approximately 4 mastoid cells above and posterior to the knee of the sigmoid sinus, at the level of the mastoid antrum. The pain persisted for two or three days, and examination showed the same condition as previously reported. The patient wanted an operation performed on the mastoid, which seemed justifiable because of the definite history and because of the roentgen-ray evidence that a few cells remained in the mastoid which had not been exenterated.

On Sept. 2, 1924, the left mastoid was opened through an incision following the scar from the previous mastoidectomy. The periostium was retracted posteriorly and the mastoid was attacked at once directly over the area that the roentgenogram showed contained cells which had not been drained previously. The purpose of this procedure was to open the cells before any other part of the mastoid was operated on, so that if fluid was encountered, it would not be complicated by oozing from a large wound. The cells that gave roentgen-ray evidence of cloudiness were immediately encountered, above the sinus and posteriorly. The cell walls were not broken down, were lined with membrane and contained a small amount of clear, colorless fluid, but not enough to collect for study.

Following this, the area previously operated on, was exenterated to make sure there were no more mastoid cells and to find, if possible, a channel leading from the cells containing fluid to the mastoid antrum. No other mastoid cells were found, and there was no evidence of a channel leading to the middle ear. The dura was not exposed, nor was the wall of the sigmoid sinus. The mastoid tip was not disturbed. The mastoid wound was closed with a drain through the lower part of the incision. A paracentesis was performed.

The entire postoperative course was uneventful. The patient gained 2 pounds (9 Kg.) while in the hospital. Subsequently, there was never any drainage

through the middle ear, and the mastoid wound was allowed to close. The patient left town on September 17. Several communications from him, the last one in December, 1925, state that he is well and has had no recurrence of his former trouble. It appears that this case was one of primary sterile secretory mastoiditis.

THE VALUE OF THE ROENTGENOGRAM IN SINUS AND MASTOID SURGERY. Presented by DR. G. HENRY MUNDT.

To obtain maximum information from the roentgenograms it is essential that they be made with regular and constant methods. The otolaryngologist should develop the ability to interpret roentgenograms in order to secure full value from them. A written report from the roentgenologist may be of some assistance, but it is a poor excuse for a shadow-box beside the patient during an examination. This is especially true in sinus work. The two main factors in reading roentgenograms are anatomy and pathology. Whether flat or stereoscopic plates are of most value is a moot question, but Dr. Mundt questions seriously whether the flat plate is not just as serviceable in the majority of cases. In mastoid films the condition of the overlying tissue must be considered, since changes in the soft parts may cast a shadow on the film simulating an involvement of the mastoid. If coalescence is watched for, there will probably be no misinterpretation. Roentgenograms of the sinus are of great value, but here, more than in the case of mastoid, close cooperation between the roentgenologist and the otolaryngologist is needed. Slight changes in a sinus shown on the roentgenogram may be of great importance when considered with other symptoms. While in the future there will be great improvement in the visualization of sinuses and mastoids, the close study of roentgenograms as they are made today is well worth the effort and time expended. Roentgenogram must be used in conjunction with the other elements of an examination in order to arrive at a diagnosis.

DISCUSSION

DR. BECK: The essayist has overlooked a recently described means of examination of the sphenoid sinus, which is very reliable. With the plate placed beneath the lower jaw and the roentgen ray passed through the occiput, the sphenoid sinus will be demonstrated in a stereoscopic way. Dr. Reeves of North Carolina demonstrated such a plate, which showed not only the sinus, but that the walls were necrotic and the sinus clouded. This method is a great contribution to the study of the sinuses. The Granger method has been made negative by many roentgenologists and rhinologists who have proved it to be without value. I do not think that the anatomy of the sphenoid sinus is the same on both sides, and Prentice of Iowa City has shown a great variety of sphenoids going into the pterygoid fossa.

I agree with Dr. Mundt that the pathologic condition in many cases is not being determined, and I think this is due to the fact that the plates are not studied with magnification. If this is done, much valuable evidence can be found in the roentgenogram.

DR. MUNDT: I hope that Dr. Beck is right and that I can project the sphenoid sinus in the way he mentioned in a group of cases. A thesis should follow the opinion of the writer, and I have tried many methods of projecting the sphenoid without finding any that was uniformly satisfactory.

A. A. HAYDEN, M.D., Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLOGY AND LARYNGOLOGY

April 20, 1927

DR. JAMES A. BABBITT, *Chairman*

NEUROLOGIC COMPLICATIONS OF THE EAR, NOSE AND THROAT. DR. T. H. WEISENBURG.

Dr. Weisenburg said that for eight years he had given lectures to students of otolaryngology in the Graduate School of the University of Pennsylvania. During that period he had seen a vast number of the conditions of the ear, nose and throat which show points of neurologic interest.

The association of optic atrophy, optic neuritis and choked disk with nasal conditions is important. Some rhinologists consider this sequence common. Others deny its frequency. Sluder considers that the optic nerve is involved by direct extension, by hyperplasia of the soft parts or by pressure following osteitis or periostitis. Not all cases can be so explained, however, for often no pathologic condition can be found in the sinus, yet surgical treatment of the sinus improves vision. Perhaps these cases are due to toxic influences. Retrobulbar optic atrophy is more common than optic neuritis. This can follow sinus infection without direct involvement of the optic nerve, probably by toxic influences. Primary optic atrophy never follows sinus disease.

Dr. Weisenburg said that he did not consider that sinus disease can or does produce a frank choked disk. Some observers have reported choking to the extent of 8 diopters. Dr. Weisenburg said that he did not believe that choking to the extent of more than 2 or 3 diopters ever occurs, this being due to optic neuritis. The procedure of operating on the sinuses for optic neuritis before a thorough neurologic study has been made is to be condemned. The brain surgeon sees many cases of tumor of the brain in which nasal operation has previously been performed. Pain in the face may be due to involvement of the fifth, ninth or nasopalatine ganglion or of the sympathetic system. Dr. Weisenburg said that his experience with pains of the sphenopalatine ganglion has been disappointing in that, while cocainization has temporarily alleviated the pain, alcholic injection of the ganglion has failed to do so. The nasopalatine pain as described by Sluder does occur, but the syndrome is uncertain. This may be due to variations in the ganglion, to which Sluder has called attention. Cushing and Frazier doubt the existence of this syndrome.

Inflammatory processes of the middle ear, sinuses or nose may give rise to thrombophlebitis of the dural sinuses, meningitis, extradural abscess and abscess of the brain. About 90 per cent of abscesses of the brain follow chronic infections. The diagnosis of abscess of the brain is most difficult. The history must be carefully considered, the clinical course followed, and frequent neurologic studies made for the detection of signs of localization.

Vestibular tests should always be made as an aid in the diagnosis of an intracranial lesion. They are not infallible, and the diagnosis should not be made from these tests alone. Vertigo may be toxic, epileptic or functional in origin. In my experience, most cases of vertigo have proved to be toxic and have a good prognosis. It is difficult, but important, to distinguish between aural vertigo and the minor types of epilepsy.

Encephalitis affecting the vestibular nuclei in the medulla may give rise to labyrinthine vertigo.

DISCUSSION

DR. WELLS P. EAGLETON: The future may be considered bright in regard to the cure of meningitis. At the beginning of the disease, all patients are curable. If at this time adequate surgical drainage can be provided, meningitis of a purulent nature can be prevented. All efforts to cure general leptomeningitis have failed.

The chronological history is of great value in the diagnosis of abscess of the brain. Chronic abscess of the temporosphenoidal lobe is to be differentiated from cerebellar abscess by (1) visual fields and (2) ventricular puncture. In lesions other than those of the pontile angle the vestibular tests are of little localizing value. These tests are of value in detecting increased intracranial pressure of the posterior fossa, trauma — especially hemorrhage — in the posterior fossa, toxic conditions and disseminated sclerosis. In all cases of vertigo, turning and caloric tests should be made. If the duration of the nystagmus is reduced, they should be considered cases of focal infection. Arteriosclerosis and sexual causes may give rise to vertigo. Dr. Ross Hall Skillern considers the toxic possibilities most important in retrobulbar neuritis. Focal infection, direct extension of infection, hyperplastic sinus conditions, toxic influences and negative pressure must all be considered. Operations providing adequate aeration and drainage must be performed on apparently normal sinuses in many cases of retrobulbar neuritis. Improvement is due to ventilation and to relief from toxemia. If there is a rapidly progressing optic neuritis, operation should be performed at once. Dr. Skillern's experience with symptoms of the sphenopalatine has been disappointing.

DR. LOUIS CLERF: I have been impressed by the fact that the larynx featured but little in the report of the neurologist. Many times a sensation of foreign body in the throat is due to the lingual tonsil. Laryngeal pain is most often referred to the ear. Phonic spasm, congenital stridor of infants, infantile laryngeal paralysis, laryngeal vertigo, nervous cough and functional aphonia all have a neurologic background. In paralysis of the recurrent laryngeal nerve, the cause of the paralysis is sought elsewhere.

DR. NATHAN P. STAUFFER: When should surgical intervention be used in purulent meningitis?

DR. GEORGE P. COATES: I should like to ask Dr. Eagleton what his procedure is in sterile meningitis without localizing signs.

DR. EAGLETON: Surgical intervention should be used when there is localized infection.

DR. WEISENBURG: Every otologist should have some knowledge of ophthalmology. He should be capable of reading eyegrounds and taking visual fields. Many times the vestibular tests show evidence of cerebellar tumor when toxic conditions, such as alcohol, lead or syphilis may be the causative agent. In all functional diseases, the investigation of sexual conditions is important.

Book Reviews

PRACTICAL OTOLOGY. By MORRIS LEVINE, M.D., New York. Price, $5. Pp. 387, with 145 engravings and 3 colored plates. Philadelphia: Lea & Febiger, 1927.

The title of this book expresses the nature of the contents well. The author has succeeded to an unusual degree in giving a concise treatise discussing the practical problems that confront the otologist. The conclusions are based on personal observations and years of careful study. The arrangement and the style of the text are the author's, and he presents his material in a pleasingly direct and convincing manner. The book will be a helpful guide to the student who aims to acquire a working knowledge of the problems as they are understood today in this special field. The conclusions throughout represent the mature results of the author's personal experience and are in accord with the best teaching. The chapters covering the suppurative diseases of the ear are especially well presented, and his discussion of the indications for operative intervention, both in acute and in chronic processes, should help to clarify the haziness concerning this which often exists.

One could point out a number of minor errors; for example, when he refers to the handle of the malleus as its long process, on page 22, and when he undertakes the designation of the several tuning forks, on page 26. One wonders just what the author has in mind when he refers repeatedly (pages 320, 326 and 332) to the loss or complete absence of bone conduction in one-sided labyrinthine deafness, since it is impossible to test bone conduction in the two ears separately. One also wonders just what he means, on page 324, when he says that perilabyrinthitis can be differentiated from diffuse suppurative labyrinthitis by an absence of the fistula symptom, and again, on page 324, when he speaks of a diffuse type of suppurative labyrinthitis without a complete loss of function.

Directory of Ophthalmological and Otolaryngological Societies

NATIONAL

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. D. Crosby Greene, 23 Bay State Road, Boston.
Secretary-Treasurer: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Place: Hotel Chelsea, Atlantic City, N. J. Time: May 21, 1927.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Chevalier Jackson, Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J. Time: May 23-25, 1927.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Burt R. Shurly, 62 W. Adams Ave., Detroit.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.
Place: Atlantic City, N. J. Time: May 26-28, 1927.

AMERICAN OTOLOGICAL SOCIETY

President: Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: D. Harold Walker, 390 Commonwealth Ave., Boston.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Ross Hall Skillern, 1928 Chestnut St., Philadelphia.
Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Book-Cadillac Hotel, Detroit. Time: September 12-16, 1927.

LOCAL

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Calhoun McDougall, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Zach W. Jackson, Doctors Bldg., Atlanta, Ga.
Place: Academy of Medicine, 32 Howard St. Time: 8 p. m., last Thursday of each month.

SECTION ON OTOLOGY AND LARYNGOLOGY OF THE BALTIMORE CITY MEDICAL SOCIETY

Chairman: Dr. Edward A. Looper, 605 Park Ave., Baltimore.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Arthur M. Corwin, 25 E. Washington St., Chicago.

Secretary: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.

Place: Grill Room, Stevens Restaurant, 16 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Frederick W. Lamb, Provident Bank Bldg., Cincinnati.

President-Elect: Dr. Walter Murphy, 707 Race St., Cincinnati.

Secretary-Treasurer: Dr. Harris H. Vail, 24 E. 8th St., Cincinnati.

Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

THE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SECTION OF THE CLEVELAND ACADEMY OF MEDICINE

Chairman: Dr. W. J. Abbott, 8314 Euclid Ave., Cleveland.

Secretary: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.

Place: Winton Hotel. Time: Fourth Friday of each month.

THE COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. David A. Strickler, Empire Bldg., Denver.

Secretary: Dr. C. Howard Darrow, Metropolitan Bldg., Denver.

Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. Ivimey Dowling, 116 Washington Ave., Albany, N. Y.

Secretary-Treasurer: Dr. L. Prescott Brown, 33 Bancker St., Albany, N. Y.

THE KANSAS CITY EYE, EAR AND NOSE SOCIETY

President: Dr. Joseph W. Kimberlin, Rialto Bldg., Kansas City, Mo.

Secretary: Dr. A. E. Eubank, 636 Argyle Bldg., Kansas City, Mo.

Time: Third Thursday of each month from September to May; the November, January and March meetings are devoted to clinical work.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Val H. Fuchs, Chaille Medical Bldg., New Orleans.

Secretary-Treasurer: Dr. Carl A. Granberry, Maison Blanche Bldg., New Orleans.

Place: Senses Hospital, Touro Infirmary, and Charity Hospital (in rotation).

Time: 8 p. m., third Thursday of each month from September to June.

THE SECTION ON LARYNGOLOGY AND RHINOLOGY OF THE NEW YORK ACADEMY OF MEDICINE

Chairman: Dr. John E. Welch, 138 W. 70th St., New York.

Secretary: Dr. Arthur Nilsen, 40 E. 41st St., New York.

Time: 8:30 p. m., fourth Wednesday of each month from October to May.

THE OTOLOGICAL SECTION OF NEW YORK ACADEMY OF MEDICINE

Chairman: Dr. J. L. Maybaum, 12 E. 86th St., New York.

Secretary: Dr. R. T. Atkins, 4 W. 53d St., New York.

Place: 103d St. and Fifth Ave. Time: Second Friday of each month from October to May.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Charles J. Coffey, 221 Grand Ave., Milwaukee.

Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.

Place: University Club. Time: 6:30 p. m., third Tuesday of each month

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.

Secretary: Dr. M. B. Selingstein, 801 Union and Planters Bank Bldg., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

THE SECTION ON OTOLOGY AND LARYNGOLOGY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

Chairman: Dr. Fielding O. Lewis, 261 S. 17th St., Philadelphia.

Clerk: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.

Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

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